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DUODENAL ULCER

A CLINICAL DISCUSSION ON THE TREATMENT AND THE COMPLICATIONS FOLLOWING GASTRO-ENTEROSTOMY, WITH TWO CASE REPORTS

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Students in the final year of their medical course are liable to consider only the changes of opinion regarding the treatment of any medical or surgical condition which have taken place in the years since they entered Medicine. They are liable to think that the present opinions given and taught today regarding Duodenal Ulcer, which have not altered much in the past six years, have been standard for some time. Am I assuming too much in saying that few of you realize what the considered opinion of the treatment of Duodenal Ulcer was, so short a time as fifteen or twenty years ago? To you this may seem a long time, yet to your Clinicians it is a comparatively short period during which the surgical opinion regarding the treatment of Duodenal Ulcer has completely changed, both as to the indications for operation and the procedure to be carried out. And don't be so sure that the opinions of today may not change tomorrow. The Internist and the Surgeon are far from happy in the absence of a better known treatment, to submit the patient with Duodenal Ulcer when surgery is indicated, to such a radical procedure as partial Gastrectomy, to remedy what seems to be so simple a condition. There are those among you who in future may solve this problem and give us the final answer to the treatment of Peptic Ulcer or Duodenal Ulcer to which this short paper refers.

Some twenty years ago, the Surgeon gave it as his opinion, when diagnosed, that the treatment was surgical and that the operation to be performed was Gastro-enterostomy. During the years following, there is a voluminous literature, published results by the Surgeon, and numerous papers by the Internist, who did not subscribe to the above opinion. May I refer briefly to a few of the contributions to the Surgical literature—the writings of the late Lord Moynihan, Patterson, and Sir James Walton in England, and the Mayo Clinic on this Continent. What now appear to have been extravagant claims as to results of Gastro-enterostomy were published:

85% cures,
12% well with some symptoms,
3% serious complications.

Time convinced the surgeon that the disappointments were much greater and the figures quoted misleading. Whilst the serious complications may occur as early as six months, in many it is considerably later than two years and as late as ten years after Gastro-enterostomy. It is possible cures were reported too soon and not after a sufficiently long follow-up. There are those who will say the cures following Gastro-enterostomy were never greater than fifty per cent. Even though this low figure be correct it is only fair to remember that during these years Gastro-enterostomy became an operation mastered by and performed by many. One must admit a good number of operations may have been performed where unnecessary, (diagnosis not established) and possibly not too well done, (stoma

too small and not properly placed).

In view of our present idea of Duodenal Ulcer and its treatment one wonders why the results were so good. Before, all cases of Duodenal Ulcer when diagnosed were considered by the surgeon as surgical, and at operation a Gastro-enterostomy was performed. Now we agree with the Internist that all cases are to be regarded as medical and receive medical treatment—that the condition is to be considered surgical only when medical treatment fails, i.e. after repeated haemorrhages or in the presence of obstruction—and that the surgical treatment, except in some cases, is Gastric Resection and not Gastro-enterostomy. There are surgeons today who state that in all cases the operation to be performed is Resection and that they will never again do a Gastro-enterostomy. I cannot subscribe to that opinion and I still consider Gastro-enterostomy the operation suitable in the patient with definite organic pyloric obstruction, and in the elderly patient with relatively low acidity. Published results have always been uniformly good when Gastro-enterostomy has been performed in cases of Pyloric Obstruction. The mortality in Resection for ulcer is generally conceded as four percent, and for Gastro-enterostomy one percent. In the older age group this four percent probably will be higher. We are following cases through our Gastro-intestinal Follow-up Clinic who fall into these two groups, on whom Gastro-enterostomy was done, the obstructive and the elderly, whose results to date, nearly three years after operation (admittedly too short a time) are very gratifying, and up to the present see no reason to submit these patients to the more major procedure with a higher mortality.

It must be admited that the obstructed cases make a very small group—that much the larger percentage of cases fall into the group where Gastro-enterostomy should not be done—yet for years this larger group without obstruction, with high acidity, high motility—when operated upon had Gastro-enterostomy performed, which we now consider was the wrong operative procedure, and which should have had partial resection. In view of this I repeat again—one wonders why the results were as good.

As stated above the surgeon has changed his opinion, and agrees that surgery is not to be recommended when first diagnosed—he has admitted that Duodenal Ulcer is a medical disease and should be so treated. Internists and Surgeons agree that operation is indicated when medical treatment fails or complications present themselves, viz. adherence to head of Pancreas or Gall-bladder, or repeated haemorrhages, or organic pyloric obstruction.

Is it possible there still can be disagreement as to when medical treatment fails? Who is to be the judge? The late Lord Moynihan stated he only operated on cases which had been cured many times medically. The pendulum may swing too far backwards, and it is, I think possible today that there are cases which should have been referred for operation earlier.

I wish now to refer briefly to our present position—that the operation in most cases should be Gastric Resection. Are we satisfied with our results? Can complications follow Resection? The answer is "Yes", and when stomal ulcer does follow resection, the surgeon is confronted with a condition much more formidable than when it followed Gastro-enterostomy. Then he could still do Re-

section. What can he do now? The treatment of complications following partial Resection will not be discussed in this paper.

For years most surgeons practiced doing Resection by the posterior route—the Polya operation—modification of Bilroth II. As early as 1926 the late Lord Moynihan advocated the ante-colic isoperistaltic anastamosis in his revised classical two volume books—Abdominal Operations. Later Balfour (Mayo Clinic) recommended the ante-colic anti-peristaltic anastamosis with entero-anastamosis.

The Lahey Clinic for two or three years have advocated and performed only the ante-colic anastamosis, not for reasons advocated by Moynihan or Balfour—but because it is a satisfactory operation and more easily performed, and less time-consuming, and in case of complication, the complication can be more easily and safely dealt with. That complication is stomal ulcer, and in a recent article Lahey reports seven cases of stomal ulcer proven at operation following a previous gastric resection. Stomal Ulcer can follow resection.

The changes in opinion as to the management of Duodenal Ulcer already referred to have not suggested any change in our ideas of the treatment of the acute perforation. Some twenty years ago ambitious surgeons recommended, in patients with acute perforations, seen early, in the younger age group, at the same time as closing the perforation, doing a posterior Gastro-enterostomy. Today, of course, it is obvious that if the type of case which perforates is the patient who should have Resection, Gastro-enterostomy should not be performed. The case which perforates is invariably the case requiring Resection.

A time later, a group of surgeons in Vienna advocated under similar conditions partial Gastrectomy, with a mortality of six per cent. Surely there can be no justification for such a procedure. In patients under sixty years with perforations of Duodenal Ulcer operated on at the Western Hospital under six hours, we have found our mortality in a five year period is only one-half to one per cent. What justification is there to increase the mortality in this group to six per cent.—and none of these cases were treated with the sulphonamide drugs. Therefore, the treatment today for perforations is the one we are so pleased to hear from students—"Get in quickly—get out quicker."

The main and serious complication following Gastro-enterostomy for Duodenal Ulcer is Stomal Ulcer and complications following Stomal Ulcer. I leave it to you to refer to the literature to refresh your memories as to the details—symptoms, etc., of this complication. All I wish to say here is that it may occur six months to ten years following Gastro-enterostomy—that the symptoms may simulate somewhat the original attack of Duodenal Ulcer—that the symptoms may be haemorrhage alone, but usually indigestion and pain with or without haemorrhage—that it is much more resistant to and more difficult to control by medical treatment than the original ulcer—that the patient may have a stomal ulcer (which is on the jejunal side always), and barium meal be negative—that the patient may have symptoms for a considerable time before the Radiologist can tell you that there is any suggestive distortion of the afferent or efferent loops and even when there is distortion of either afferent or efferent loop close to the stoma, in most cases he will not demonstrate a crater. From the history of the patient before his Gastro-enterostomy was performed one can assume that he or she was the type where

Gastro-enterostomy should not have been performed, and with the return of symptoms, in the absence of the Radiological evidence, he should strongly suspect that stomal ulcer is present.

Two case reports are given to illustrate this complication and its management. Stomal Ulcer and Gastric Jejunal Colic Fistula.

Case 1. Stomal Ulcer.

Mr. C. Age 70. This patient had suffered from attacks of indigestion from Duodenal Ulcer since 1935. In 1938 diagnosis was confirmed by X-ray. On March 12th, 1939, he had a severe attack of pain in the upper abdomen which was considered to be perforation and he was admitted to the Western Hospital and operated upon. The patient had a Duodenal Ulcer with definite scarring in the first portion of the duodenum. There was no perforation yet the Surgeon did a Gastro-enterostomy. The same day the correct diagnosis revealed itself—Pneumonia of right lower lobe. He recovered. This case emphasizes an interesting and most important point. It is always imperative regardless of findings by history or examination to treat any patient for the condition which brings him to Hospital. Making all allowances for mistakes in diagnosis, when this abdomen was opened and the diagnosis found to be incorrect, the abdomen should have been closed and no operation should have been performed for his Duodenal Ulcer.

In May, 1939, symptoms returned—gas and pain. For the following year it was fairly continuous with only periods of seven to ten days without discomfort. There was no haemorrhage. He was re-admitted to Hospital on August 9th, 1940. X-rays at first were negative but later demonstrated an alteration in the efferent loop with a small ulcer crater.

It would appear that in this patient he showed symptoms of and developed a stomal ulcer very soon after his Gastro-enterostomy. He had several months of relief but was re-admitted April, 1941, for return of pain. As the X-rays were negative on this admission and due to the fact that he had never had a haemorrhage, he was discharged for further trial on medical treatment.

Symptoms returned in November, 1941, and in December he had tarry stools. He was re-admitted later to Hospital. The X-ray was very suggestive of stomal ulcer.

He was operated upon in February, 1942. One found the ulcer to be present. The anastamosis was undone. It was necessary to resect a portion of the jejunum. Radical partial gastrectomy was performed, ante-colic — anti-peristaltic — long loop—no entero-anastamosis. His recovery was uneventful and seen recently in Follow-up Clinic is quite well and free from symptoms.

Stomal Ulcer and Gastro-Jejunal Colic Fistula

This new ulcer as mentioned before is invariably located on the jejunal side of the anastamosis. The anastamosis in posterior Gastro-enterostomy is performed through the transverse meso-colon between branches of the middle colic artery. It is not difficult to visualize the proximity of this ulcer to the colon, and through its activity and repeated exacerbations of acute flare-ups, to understand how it may become adherent to the colon and ultimately result in the formation of a Fistula. This complication has always been considered a most serious one, and the operation for its relief accompanied by a very high mortality. Once established the patient has a distressing new symptom —frequent stools and later foul breath

and even faecal vomiting. Previously it was thought that the diarrhoea was due to the direct passage of content from the stomach to the colon and thence to the rectum. The X-ray investigation of these cases is interesting. Only with difficulty can the Radiologist force barium through the fistula. Yet by the barium enema the stomach fills readily. It is now thought that the explanation for the diarrhoea is that faecal material in the jejunum is responsible for excessive peristalsis and the frequent movements. This is borne out at operation, where the colon is first defunctioned by an ascending colostomy and the patient almost at once ceases to have diarrhoea.

The reader is referred to the article by Pfeiffer—S.G.O. Feb., 1941—where this is discussed in detail and where ascending colostomy to defunction the colon is the first operative procedure. This is followed some time later by undoing the anastamosis and fistula, closing the fistula in the colon, closing the jejunum and resecting the stomach. This appears to be a great advance in our knowledge of the treatment of this complication and converts a formidable procedure with high mortality into a simple one.

Case 2. Gastro-jejunal Colic Fistula

Mr. F. Age 38. He suffered from indigestion very early in life. In 1923 (age 20) he was operated upon in Saskatoon for perforated Duodenal Ulcer. He was advised later to have a Gastro-enterostomy. He went to the Mayo Clinic where he was advised to have focal infection (teeth and tonsils) removed, and put on a diet. In 1924 symptoms returned in spite of the fact that he cooperated with the advice given. From 1924 to 1927 he carried on, taking considerable alkali for relief. In 1927 his symptoms became more pronounced and he submitted to operation (Gastro-enterostomy). In six months symptoms returned and from 1928 to 1939 he disregarded advice and took alkali for pain. He had no haemorrhages. It is apparent that during this time he was suffering from a stomal ulcer, for in 1937 he devloped frequent stools. From 1937 to 1941 he had diarrhoea, worse at night. The stools were watery. There was no difference with liquids or solids, when food was taken or how cooked. There were no haemorrhages. At times his breath was quite offensive but he had no faecal vomiting. He was admitted to the Western Hospital September 30th, 1941, barium enema readily filled the stomach and established the diagnosis. He declined treatment but returned January 5th, 1942. On January 10th, 1942, first stage operation was performed—Ascending Colostomy. Examination of the stoma and fistula revealed the ulcer—the fistula and considerable congestion of all structures in the region of the anastamosis, and a peculiar muddy water colour of the jejunum distal to the anastamosis. Following this operation the diarrhoea ceased.

The second operation was delayed by a haemorrhage from the ulcer and was not performed until March 7th, 1942, at which time the patient's condition had markedly improved. The anastamosis was undone. The colonic fistula was closed—the jejunum was closed—the stomach was resected and the jejunum anastamosed with a long loop (Anti-peristaltic) without entero-anastamosis. Recovery was uneventful. On May 19th the colostomy was closed and he was discharged from Hospital May 28th, 1942. Last seen October 8th, 1942, in Follow-up Clinic—he felt fine, had gained considerable weight and had no gastric symptoms.

ANO-RECTAL PAIN: CAUSES AND TREATMENT

A SYMPTOM

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I propose, in this paper, to discuss the more common surgical conditions of the Ano-Rectal region in which the predominant symptom is pain. My remarks, based entirely on personal experience, are directed to the graduating year in medicine, and to those now completing an abbreviated internship to join the armed services.

Anal Fissure

Because it is the most frequent cause of pain in this locality, we shall consider first, Anal Fissure, the precursor of which is thrombosis of one of the tributaries of the inferior haemorrhoidal veins *just above* the anal orifice. (Figure 1.)

A diagram showing the distribution of the haemorrhoidal veins: the superior haemorrhoidal, a tributary of the portal vein draining the rectum and upper part of the anal canal via the columns of Morgagni; the middle haemorrhoidal assisting in the drainage of the lower rectum and upper part of the anal canal, passing along the lateral ligaments of the rectum to the internal iliac vein; the inferior haemorrhoidals draining the anal canal up to the pectinate line and receiving tributaries from the *external* haemorrhoidal plexus, situated under the skin at the anal orifice; these drain into the internal pudic and so to the internal Iliac. The lining of the anal canal below the pec-

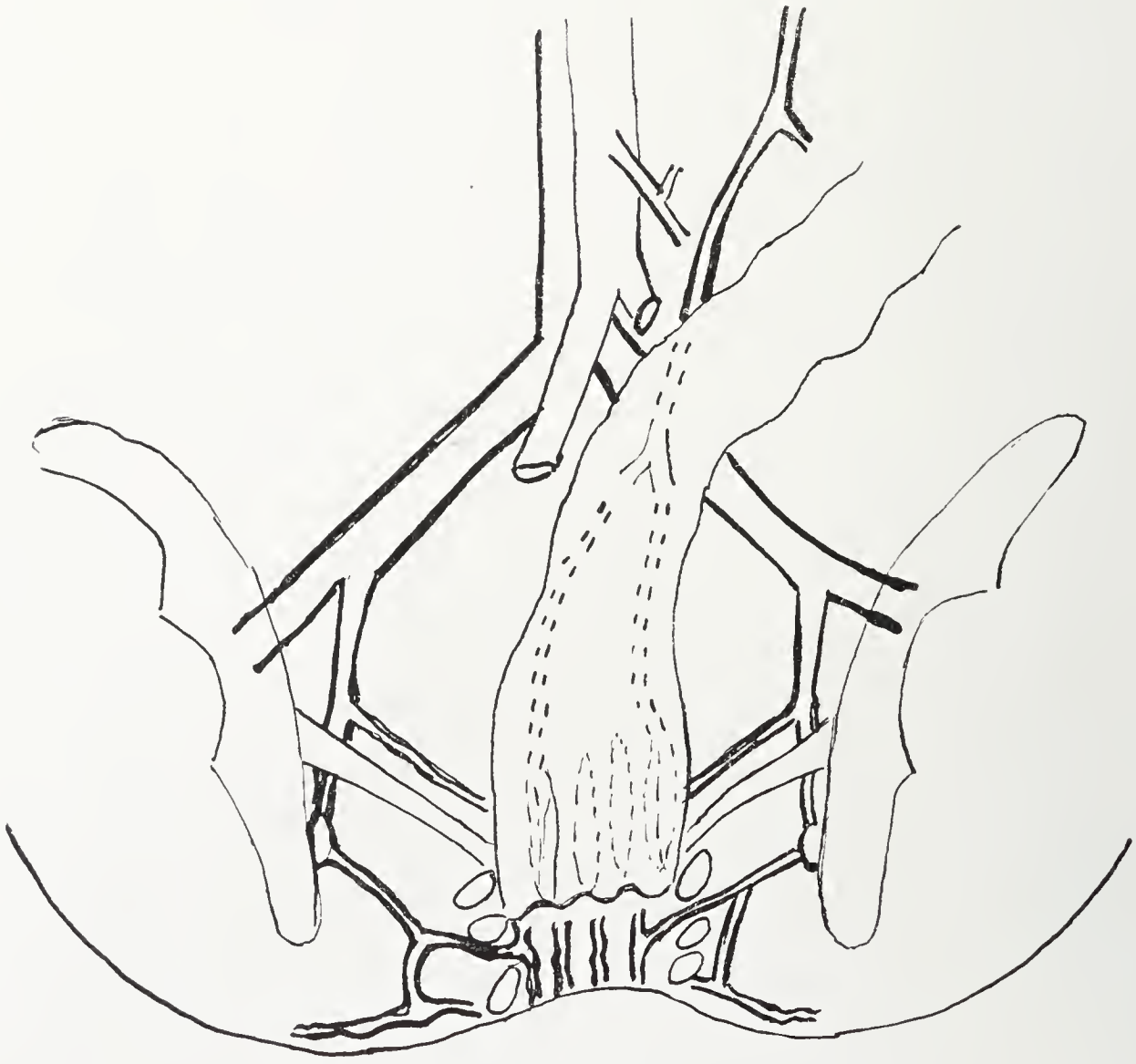

FIGURE 1

tinate line is firmly adherent to the underlying tissues, especially in its posterior quadrant, therefore, when the longitudinally disposed veins become thrombosed, or rupture into the tissues, there is little room for expansion and pain is acute, because this is the most sensitive part of the body. (Figure 2—Top.) The condition may subside leaving a fibrous ridge, but if this extravasation ruptures spontaneously on the surface, infection is sure to occur. This is the beginning of an anal fissure, (Figure 2—Centre). actually a longitudinal ulcer, the base of which is the posterior wall of an inflamed vein. The ulcer never extends beyond the pectinate line because that is the upper limit of the veins draining that portion of the anal canal. If our conception of the pathology is correct, the successful treatment is excision of such an ulcer. No other form of treatment will result in permanent cure. As the condition is always followed by anal spasm, forcible dilation under local or general anaesthesia is very tempting but will give temporary relief only. Local infiltration of the ulcer with neupercaine or other anaesthesia produces the same temporary effect.

The excision includes the fissure, usually an adjacent inflamed papilla, and the so-called "sentinal pile", if present, as well as a small portion of perianal skin, the resulting wound being pear-shaped. Under no circumstances should the excised edges be apposed. The margins of the incision are tacked down to the superficial fibres of the external sphincter, by means of interrupted plain No. 1 catgut. (Figure 2—Bottom.) It is evident that healing will progress from within outwards, and that the last part of the wound to heal will be on the skin surface. This is a principle which should always be adhered to in dealing with wounds in this region.

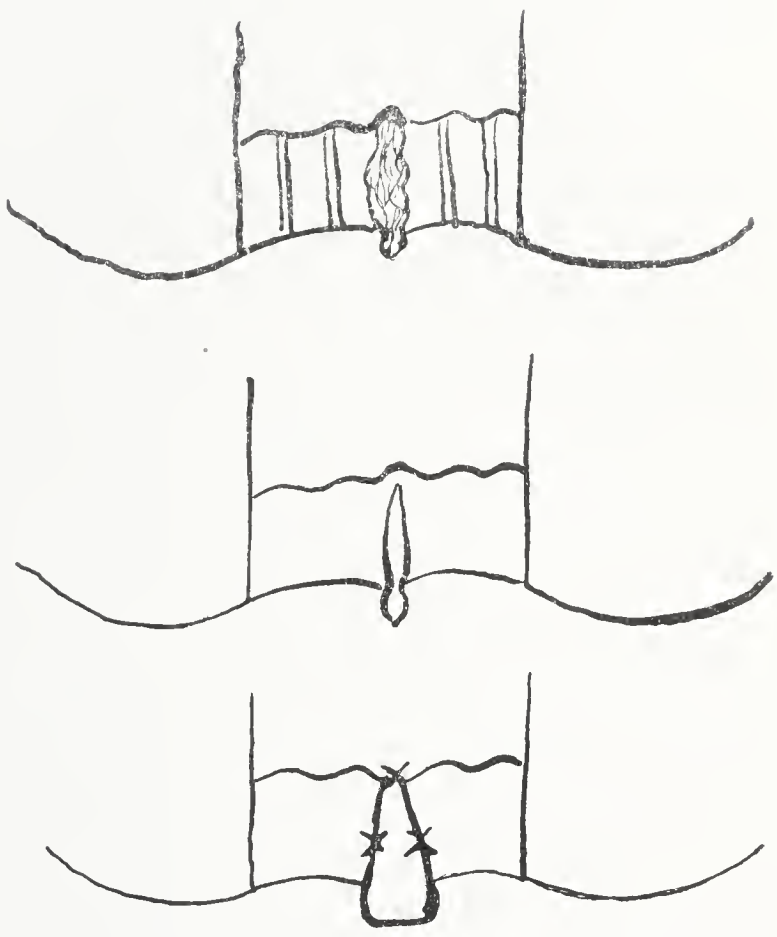

FIGURE 2

Post-operative care consists of adequate sedatives, warm boracic compresses q.4.h., sitz baths (normal saline) t.i.d. Fluid diet to soft diet second day post-operative. If home conditions permit, patient is allowed to leave hospital at the end of one week; local treatment should be continued up to the end of the third week. Digital rectal examination begins at end of the first week, and should be repeated every three or four days, to prevent the wound edges from becoming adherent. It is rarely necessary to carry out this procedure beyond the end of the third week, when a narrow, painless scar has replaced the ulcer.

Thrombosis of External Haemorrhoidal Veins

Another very painful condition is thrombosis of external haemorrhoidal veins *just outside* the anal orifice. The

onset is acute, and on examination a bluish swelling is seen at the anal orifice. The treatment consists of local infiltration with novocaine, 2 cc. of 2%, and removal of the clots through a radial incision. The wound edges are not sutured. Oozing is arrested by the application of some astringent such as silver nitrate. The wound heals rapidly on frequent bathing with normal saline. The procedure can be carried out in the examining room and the patient may continue with his work. If the condition is not treated the blood clots may become partially liquefied and infected, leading to a small peri-anal abscess, or the clots may be discharged spontaneously through a break on the skin surface in a few days.

Strange as it may sound, this condition is rarely accompanied by Internal Haemorrhoids.

Irreducible Anal Prolapse with Thrombosis of Internal and External Haemorrhoids

The history of such a case is that the individual has been suffering from internal haemorrhoids; there is a prolapse on the slightest provocation which becomes increasingly difficult to reduce, and finally becomes irreducible. (Figure 3) Stasis occurs, followed by thrombosis of internal and external haemorrhoidal veins and later their accompanying arteries, leading to strangulation and gangrene, the appearance to the inexperienced being that of a malignant growth. The case is rarely seen early enough to attempt reduction. I have come to adopt a more conservative form of treatment for this type of case than that generally recommended. Attempts at reduction of the prolapse at this stage are not only futile but injurious. I cannot see that any useful purpose is accomplished by making multiple radial incisions, as very little serum and blood exudes from the cut surfaces; furthermore, the blood clots are multiple, and adherent, so that any attempt at enucleation leads to further tissue damage and haemorrhage; in short, the condition so treated is made worse. For much the same reasons, I am against multiple excisions. For some time now I have

FIGURE 3

managed this type of case as follows: they are put to bed, the foot of which may be elevated to assist drainage: local application of cold, saturated magnesium sulphate compresses, soft diet, saline or other non-irritating enemata, instead of cathartics, and, if they have not had any

about ten days, to return for excision of haemorrhoids in about three weeks.

Thrombosis of Internal Haemorrhoids High up in Anal Canal

(Figure 4) There is always an associated extravasation of blood. The

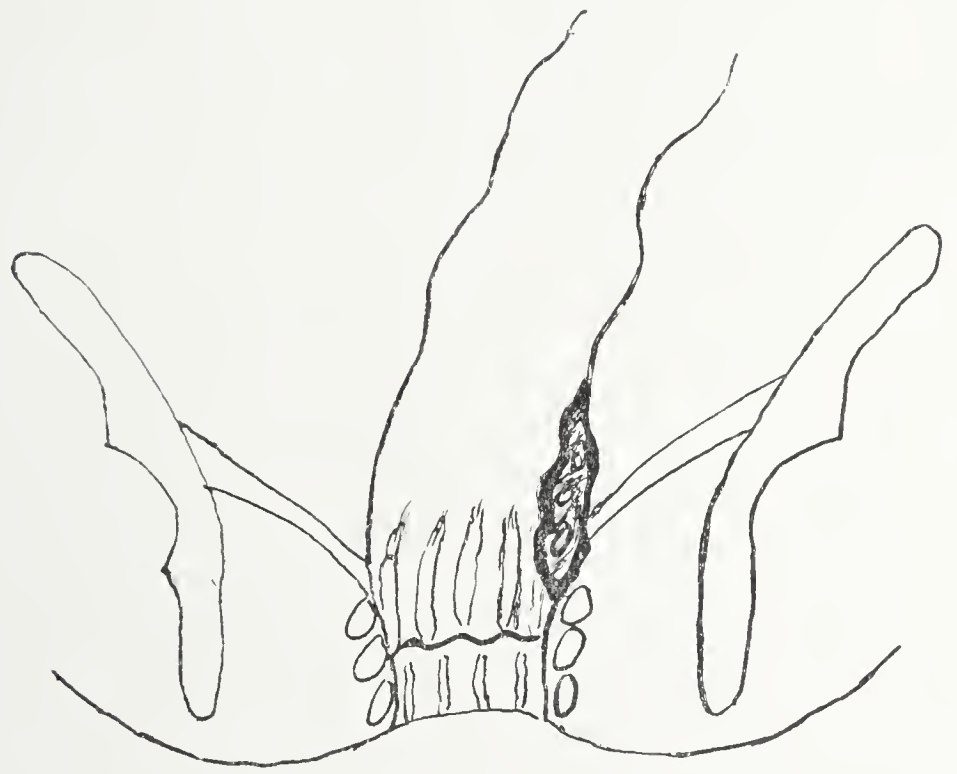

FIGURE 4

sleep for a couple of days, hypodermics of morphia are permissible for twenty-four hours. Improvement under this treatment is immediate, the swelling subsides and the patient is allowed home in

development is acute and very painful, and may follow straining, as in lifting heavy weights. It is occasionally found in cases of high blood pressure. The condition may subside, leaving a firm

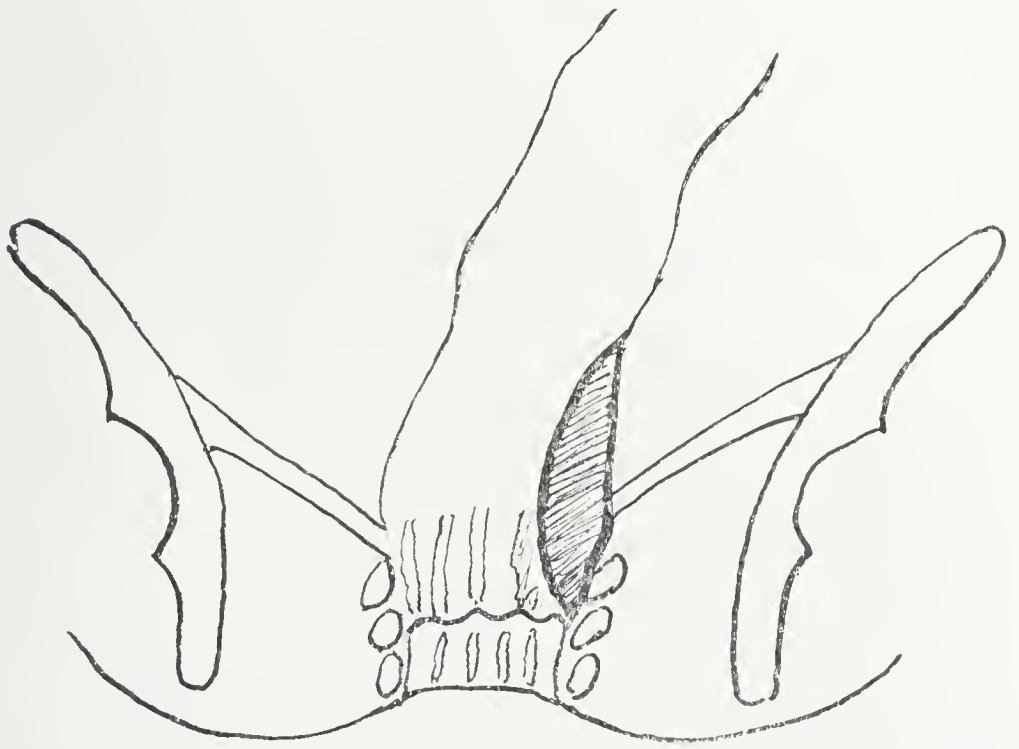

FIGURE 5

swelling which is readily felt on digital examination, and may be mistaken for a carcinoma. Most cases are afebrile, and when there is an elevation of temperature, it is very slight. I have been impressed with the common complaint of throbbing pain, which is sufficient to keep the patient awake, and seems out of proportion to the degree of pathology found. The condition responds favourably to low rectal irrigations of warm saline, and subsides in about ten days. Once the diagnosis is made, I do not advocate repeated digital rectal examinations, as there is the danger of dislodging an infected thrombus. If suppuration occurs, a painful submucous abscess develops which usually ruptures spontaneously into the lower rectum. (Figure 5) If an incision is necessary, it should always be made on the bowel side where it points.

Ischio-Rectal Abscess

(Figure 6) In over ninety per cent of cases the condition is secondary to an infection in an anal crypt. It may, however, result from direct extension of an adjacent neoplasm in the lower rectum, from ulcerative colitis or other ulcerations of the rectum, or it may arise from a degenerating neoplasm in the ischio-rectal fossa itself or from osteomyelitis of the pelvic bone, or from a puncture wound on the skin. It may also arise as a direct extension of an ulceration in the vaginal wall. Whatever may be the source of such an abscess, it should be incised immediately under spinal anaesthesia in the manner shown in (Figure 6). Three convex incisions are made and the central portion is removed, thereby "de-roofing" the abscess. The cavity thus created is probed gently with the gloved finger, to ascertain the presence of deep pockets. It may be necessary to enlarge the original incision because, on no account, should the outlet be narrower than the deeper portions; in other words, a properly opened abscess should be funnel-shaped. After all bleeders have been suture-ligated, the cavity is loosely packed with gauze soaked in cod liver oil ointment, to which a few grains of sulphathiazole powder have been added. Post-operative care consists of local application of warm boracic compresses and sitz baths, the packing being allowed to soak out gradually. It should be removed completely by the third day. The wound is never repacked, as this would interfere with proper drainage. Complete healing occurs in from three to six weeks; sitz baths

FIGURE 6

and local applications being, of course, continued during this period.

During the later stages of healing it is our concern to ascertain the possible cause of the abscess; these were mentioned at the beginning of the previous paragraph. An X-ray film of the pelvis will rule out the possibility of an ostrogenic sarcoma or osteomyelitis. A proctoscopic examination will rule out the presence of ulceration in the rectal mucosa or neoplasm. An offending anal crypt with an enlarged adjacent papilla would point to the origin of a fistulous tract.

In spite of adequate treatment, about twenty percent of abscesses will develop a Fistula-in-Ano. Most of these originate from the pectinate line; a very small number owe their origin to ulceration of the lower rectal mucosa, or from an ulcerating neoplasm in the lower rectum.

Occasionally, in opening such an abscess, one may be fortunate enough to trace a thrombosed vein into the anal canal to an anal crypt, the origin of the infection. In such cases, the tract should be opened up to the skin surface thus saving the patient a further operation for fistula.

Pelvi-Rectal Abscess

A pelvi-rectal abscess (Figure 7) may originate from infection of the prostate, seminal vesicles or upper part of the vagina, a perforating carcimona or from ulcerative colitis. Unless it points in the ischiorectal fossa, it should be opened from the bowel side.

Retro-Rectal Haemorrhage

(Figure 8) Spontaneous or as the result of an injury. Pain is intense. Treatment consists of immediate removal of blood clots through a curved incision to one or both sides of the ano-coccygeal ligament, which is preserved. The space is loosely packed for twenty-four hours then the packing is gradually withdrawn. Like all large haematomata if it is not evacuated at once, it is sure to become

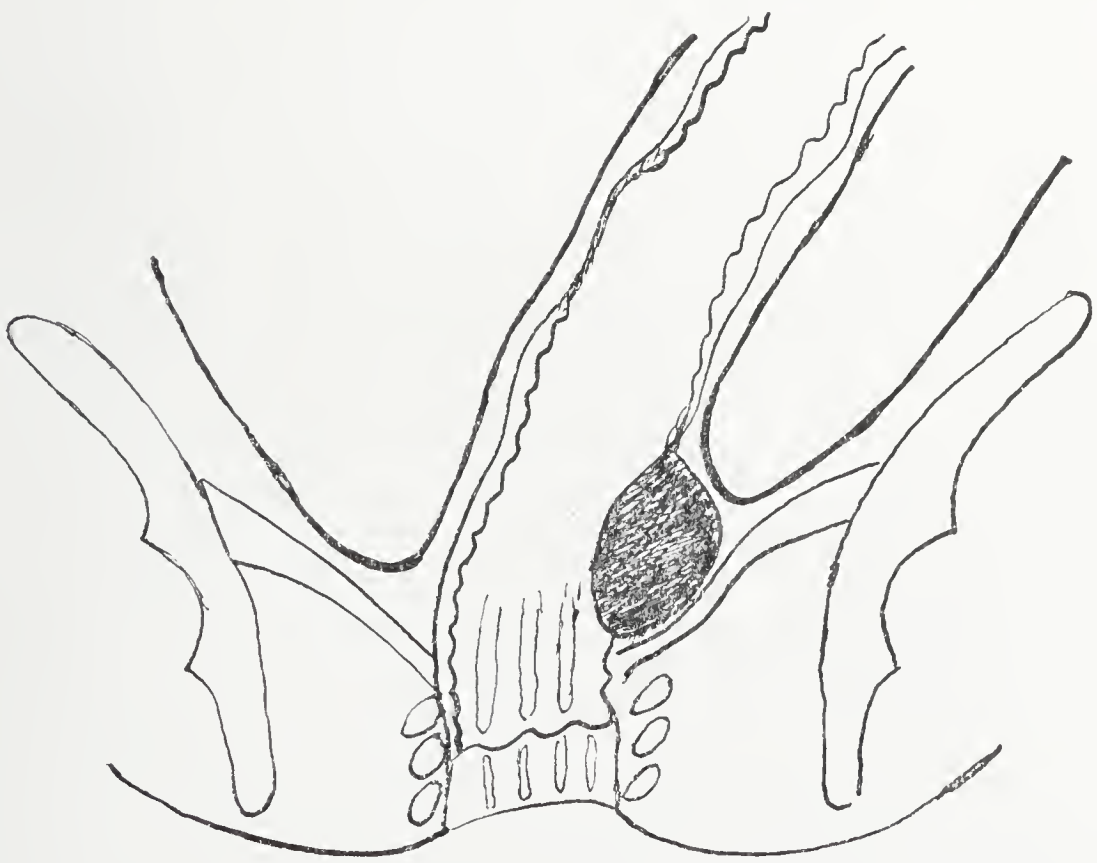

FIGURE 7

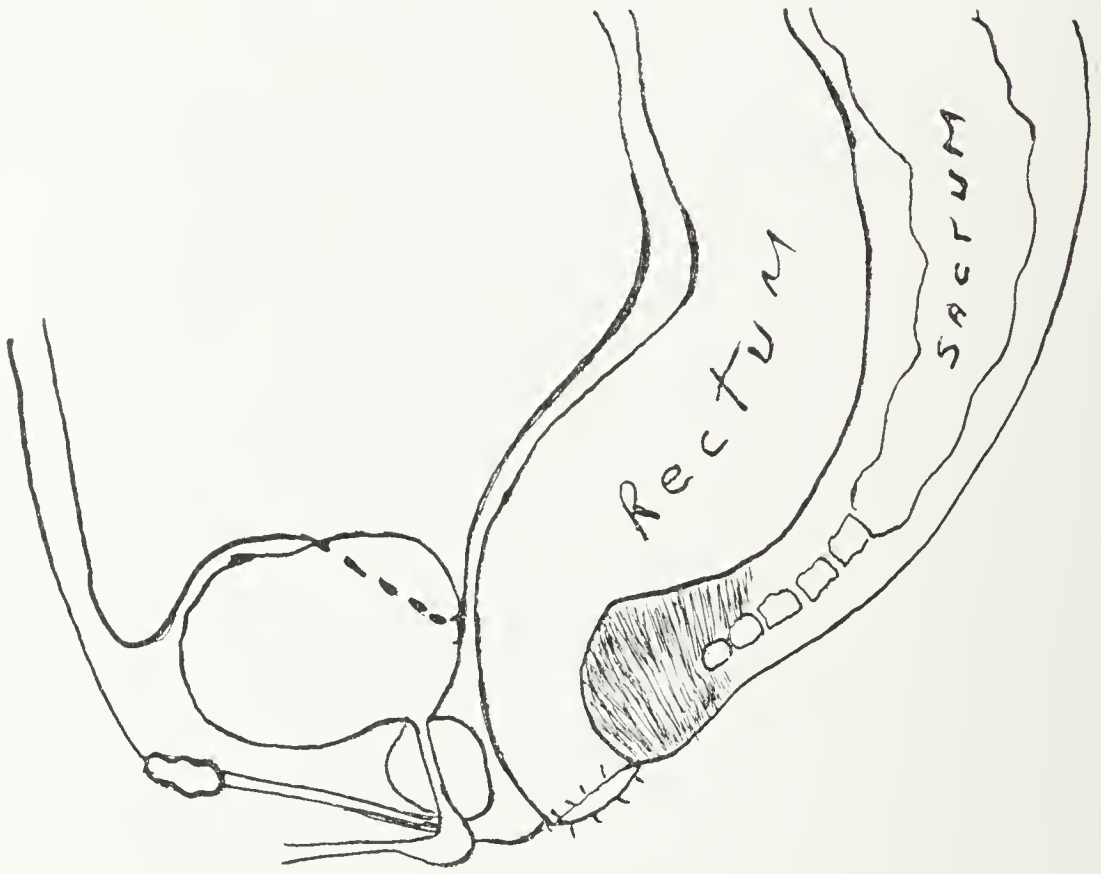

FIGURE 8

infected and result in prolonged convalescence.

Pelvic Abscesses

Pelvic abscesses which are felt on digital rectal examination, should be incised from the rectal aspect or in the female, by a posterior colpostomy.

Pilonidal Abscess

Incision and excision when the infection subsides.

Faecal Impaction

A condition frequently overlooked in bed-ridden patients. Incidently, it is a common cause of pyrexia in a bed patient who has not had an abdominal operation. The surgeon may be disillusioned by the fact that the patient, in spite of rectal impaction, may develop spurious diarrhoea or even rectal incontinence.

Neoplasms

A CARCINOMA in the lower rectum is a frequent cause of acute pain in the lower back, the rectum or the region of the symphysis, occasionally referred along the sciatic nerve to the back of the thigh or hip and for these reasons may be mistaken for sciatica or arthritis.

A TUMOUR in the ischio-rectal fossa or sacro-coccygeal region, usually easily felt through the rectal wall, may give rise to the same symptoms.

Ulcer

Another very painful condition occasionally met with in this region is an ulcer in the lower rectum or anal canal, in a case of ulcervative colitis or tuberculous colitis.

Fracture of the Cocoyx

(Figure 9) Contrary to general belief, fractures of the coccyx are not common. They are *not* produced by a fall on the back or in a sitting position or on a *flat* surface. The fall must be on the corner or edge of such an object as a box. It is important that the workman tell definitely how the accident occurred. Pain on defecation is complained of but this is

not limited to fracture of the coccyx. The patient, when sitting, if not malingering, will choose a flat, firm surface. Sitting on a soft cushion will cause pain because it is forced up against the tip of the coccyx. Examination consists of a bimanual examination of the anal canal and lower rectum, pressing against the anterior surface of the sacrum and coccyx on the bowel side and on the skin surface

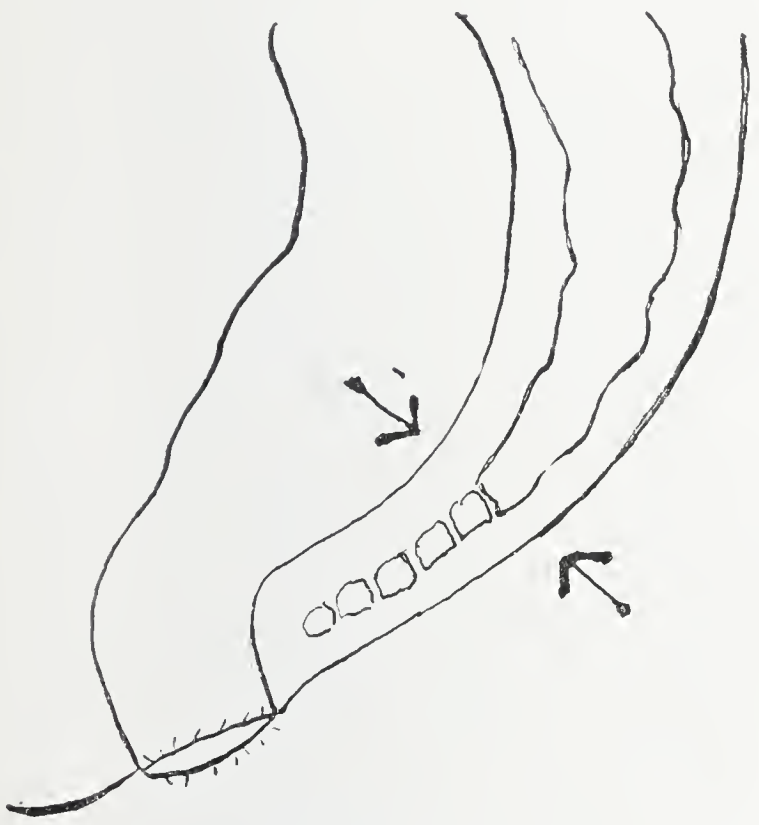

FIGURE 9

with the fingers of the other hand. It is best to begin the examination as high as possible and gradually descend upon the suspected level of injury. If there is a fracture, acute pain will be felt over the fracture site, confirmed by manipulation of the distal segment, also (and this is important) by pressure over a corresponding area on the skin surface. X-ray films are considered last and only as confirmatory evidence. Most fractures, when seen, will be found through a lower sacral vertebrae, or across the sacro-coccygeal joint. These fractures, in most cases, will respond to rest, soft diet, and sufficient cathartics to ensure one or two daily bowel movements. Union occurs in about six weeks: if, at the end of this period, symptoms still persist, it will be time enough to consider excision of the lower fragment.

Coccyodynia or Coccygodynia

The definition given in Dorland's Medical Dictionary for the above synonymous terms is "pain in the coccyx and neighbouring region due to rheumatism, neuralgia or injury to the bone." As you see, there is considerable liberty allowed the interpreter of such a definition. It is unfortunate that these terms ever found a space in our medical dictionaries. Frankly, I have never seen a case upon whom I could conscientiously pin such a label. It is the duty of the surgeon to rule out any possible local reason for the pain complained of. If this is negative, he should then refer the case to a neurologist or psychiatrist instead of removing the coccyx.

OSTEOCHONDRITIS DISSECANS

HERBERT J. SULLIVAN, M.D.

From the Orthopaedic and Fracture Service of the Toronto General Hospital.

Osteochondritis Dissecans is a joint derangement characterized by a complete or partial separation of an area of articular cartilage with a varying amount of subchondral bone.

Konig, in 1887, first described the condition in the following words, "Without an injury there separate from the joint ends, fragments of varying size in consequence of a process as yet unexplained; their bony surface becomes covered with a dense connective tissue containing cartilage cells". Eighteen years ago he gave the condition its present name but it still travels under aliases, e.g. osteochondrosis dissecans, bone necrosis, Paget's necrosis, osteochondrolysis traumatica, malcopathia of the joints, etc.

Since 1926 there have been 23 cases with this diagnosis on the Public Wards of the Toronto General Hospital. Three of these cases have been eliminated from this discussion because at operation little or nothing suggestive of osteochondritis dissecans was found.

Etiology. In keeping with the best medical traditions in dealing with diseases of unknown etiology, the list of theories is long, many of them with little foundation.

1. Trauma — Most authorities agree that trauma plays a definite role either as a direct sudden blow or as a succession of repeated minor incidents. The most interesting theory was suggested by Rieger who believes that trauma to the ends of the long bones may give rise to minute fat emboli which block the blood vessels, causing an area of asceptic necrosis which eventually separates. In our series, 6 had a clear cut history of trauma, 9 had an indefinite history and 5 gave no history of trauma.

2. Congenital Bone Predisposition. It was stated to support this theory that it was common for several joints in the same individual to be involved. This is rarely reported except when hip joints are both involved. In our series, none had more than one joint involved.

3. Low grade infection in bone produces softening which may be the earliest stage in death and separation. However, osteochondritis dissecans is a disease of young healthy adults.

4. Gonadtrophic theory. There is no proof either for or against this.

5. Upset calcium metabolism.

It is generally accepted that the cause of the necrosis and subsequent separation of the articular cartilage is due to interference of some sort with the blood supply.

Sex: Of the 20 cases to be discussed 15 were males, 5 females, which is the usual sex distribution.

Age: Varied from 15 to 45 years, but that it is clearly a disease of young adults is seen in the average age of 23.4 years.

Site: (A) *Knee*

The knee joint is involved in 85% of cases of osteochondritis dissecans. In this series in 15 out of 20 (i.e. 75%) it was involved. The area of the articular car-

tilage affected is almost invariably the lateral aspect of the medial femoral condyle, i.e. the part facing the intercondyloid notch. This constancy of position has been associated with an unusually long medial tubercle of the spine of the tibia (1).

In one of the cases reported below it was the articular cartilage of the patella which was involved. This is very rare.

(C) *The Elbow*

This joint was affected in 3 cases, one of whom was a baseball pitcher, one was a javelin thrower and one just fell. All 3 cases involved the right elbow. George Bennett (3) found that the lesion was very common in baseball pitchers and, on reviewing the literature, he found that the right elbow was affected in 14 cases, the left in but 2 (possibly these were south-

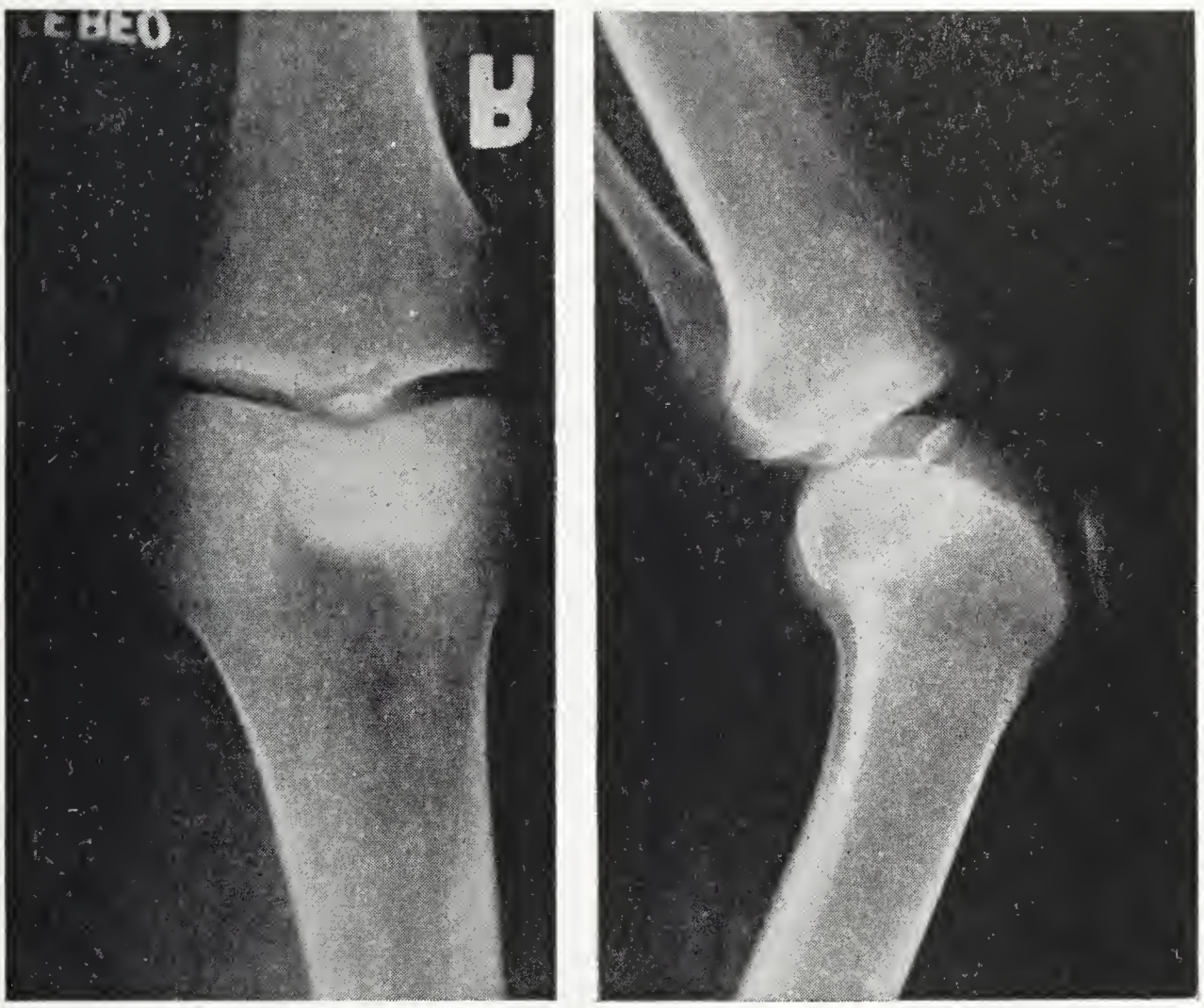

Fig.1—Osteochondritis Dissecans affecting knee-joint. Case No. 1.

(B) *Hip*

In this series there are 2 cases but there are 26 cases in the literature in which the hip was involved. When such is the case it is bilateral in 30-35% (2) of cases. It is believed to represent an end stage in morbus coxae senilis. The first case of osteochondritis dissecans of the hip, in which surgical intervention with a successful result was made, was reported from this hospital (1) in 1928.

paws). He removed a loose body from the elbow of one pitcher in 1925 and the athlete continued his profession successfully for 15 years without any recurrence of his disability.

(D) *The Ankle*

No cases in our series involved the ankle. In the literature osteochondritis dissecans involved the ankle more frequently than the elbow and almost as fre-

quently as the hip (20 cases). A cause for this discrepancy was suggested by Mensor (4) who pointed out cases in which ordinary roentgenograms were nor- but in which tomography showed the typical lesion, subsequently proved at operation. One case at this hospital had a history and roentograms strongly suggestive of osteochondritis but at operation nothing abnormal was found.

(E)—Other rare sites mentioned in the literature were phalanx (5); metatarsal (6). Recently at the Orthopaedic Clinic X-rays of a case showing Freiburg's infarction of the 2nd metatarsal were seen. In this condition a piece of articular cartilage is impacted into the head of the bone.

Clinical picture can best be illustrated by presenting a brief summary of the 2 cases which I had the opportunity of studying.

Case No. 1. A.C. Female, 21 years of age. Complaint was dull, continuous aching pain over knee joint and radiating down the leg. One week prior to admission when she attempted to extend her leg, it locked and she had a severe, sudden, sharp pain localized in her knee. Since that time she had been unable to fully extend her knee. Examination revealed that the right knee was held in a position of partial flexion, the angle being 165°. There was no limitation of flexion but neither actively nor passively could the knee be extended beyond this angle. There was no effusion present. X-rays showed a small density in the intercondyloid notch of the femur the size of a small bean (see figure). On August 17th a small incision was made over the medial side of the patella. It was seen that a piece of cartilage had been separated from the lateral aspect of the medial femoral condyle but was still attached by a fibrous band. It was easily removed. Post-operative course was uneventful and the patient walked out of the hospital on the 6th day.

Case No. 2. R.B. Female, age 17. Three years ago when rising from a chair her right knee locked. Subsequently the joint was sore and swollen but the swelling subsided with a pressure bandage. This locking became more common until over the past year it locked about once a week. The patient noted that she could release the knee by feeling a small rounded body on the medial side of her patella and manipulating it. Examination revealed no pain nor limitation of movement. No particular position caused locking. However, as she was about to leave her Doctor's (Dr. W. E. Gallie) office her knee suddenly locked and she was able to demonstrate this palpable loose body.

X-rays showed a density 0.5 cm. in diameter in the suprapatellar pouch. One edge of the patella appeared sharp and clean cut. On Sept. 14th, an incision was made down the medial border of the patella with generous elongations superiorly and inferiorly. On opening the joint a flat, button like, white, shiny osteocartilaginous disc presented itself. It was attached by a fibrous pedicle to the patella proper. Another smaller bit 3 x 2 mm. was also removed. There was considerable destruction of the articular cartilage of the lateral third of the femoral condyle. The patient was allowed up on the 4th day and home on the 6th.

Pathology. Sections of the two cases discussed above were studied. The picture presented is one of degenerating bone covered by a layer of dense fibrous connective tissue or in some areas by car-

tilage. The matrix of the bone stains a dull pinkish homogenous colour and contains numerous empty lacunae. Other lacunae contain swollen, watery osteoblasts undergoing hydropic degeneration. The cartilage on the periphery showed a well marked line of demarkation in most places but in others, it gradually blended with the underlying bone.

Radiological Studies. The roentgenographic picture as it affects the hip was first described by Dr. G. E. Richards in 1928. He described the typical lesion of osteochondritis dissecans, as "a defect appearing as a sharply defined, shallow depression irregularly circular containing in its centre a button of bone". Although roentgenorgrams showed abnormal changes in 18 of our 20 proven cases, the typical lesion was not frequently seen. The abnormality visualized varies from irregular calcified areas to one case in which a benign tumor was suggested.

Treatment. Opinion almost unanimously favours early surgical intervention with arthrotomy and the removal of loose bodies. However, there has recently been published a paper reporting spontaneous healing of the lesions of this disease in the knee joint. Wiberg (6) since 1937 has compiled a series of 10 cases treated without operation and 7 have been symptomless and 2 have shown radiographic evidence of complete, spontaneous healing.

End Results. While we have had no adequate follow up, 19 of the 20 patients have never returned to the hospital with any recurrence of symptoms. Several of them have been admitted with other afflictions since their joint operations and none have had any complaints referable to their joints. The only case, who did return, was the youngest in our series, a 15-year-old girl, who 14 months after her operation was troubled with pain and locking of her knee. Another operation was performed but no more loose bodies were discovered but she had a fairly far-advanced degenerative arthritis. This was 5 years ago and she has not been seen since.

Many thanks are due to Dr. R. I. Harris for many helpful suggestions during the writing and revising of this paper; to Miss Johnstone for secretarial assistance and to Miss Poole of the Academy Library.

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RENAL CALCULI

A REVIEW OF AETIOLOGY, DIAGNOSIS AND TREATMENT

R. F. WARREN, B.Sc. '44

Renal calculi offer the student in medicine one of the most interesting, yet practical of problems. Whether the young doctor is particularly drawn to surgery, to medicine, to research or to general practice, he is certain sooner or later to be confronted with some problem related to renal calculi. After all, this is not an entity in itself. Truly it may become his immediate problem, but he knows that stones are only and always secondary to some other condition often far removed from the kidney. It may be metabolic or bacterial but he must bear in mind that besides treating the stone he must try to find the factors which led up to its initial appearance.

It is proposed therefore to outline briefly some of the types of stones that are commonly met, and to discuss some of the likely conditions and processes which are included in present day conceptions of stone formation.

Calculi may be classified into three great classes depending on their surrounding situations.

1. *Primary Calculi* are those stones which form in an acid urine and in a non-inflamatory site. In other words these stones are sterile. Those most commonly met are:

uric acid stones
calcium oxalate
sodium urate
potassium urate, or any combination of these.

Others also found in this large group of primary stones are calcium carbonate and calcium phosphate, indigo, and xanthine. These last two are so rare as to permit known examples being counted on the fingers of one hand.

2. *Secondary Calculi* are those which are formed in an alkaline urine and are associated with inflammatory processes. Usually this implies the presence of living bacteria in and around the stone. Commonly they are found to consist of:

ammonium-magnesium phosphate
lime phosphates
ammonium urate.

3. *Mixed Secondary Calculi* as the name implies are not *Pure* secondary stones but are stones built upon a primary core. The common history is that of a pure primary stone with subsequent infection and secondary deposition over the primary. Sometimes the chemicals are arranged in consecutive layerings, indicating a repetitive infective process has been at work.

It must not be supposed that stones are as easily diagnosed as this scheme might imply. In the first place the chemical composition of a stone is seldom other than a preponderance of one chemical constituent over another. There are no chemically pure stones. In each there is an organic matrix of colloid material into which organic or inorganic salts become impregnated. Nevertheless the classification is a useful one and serves as a guide when we turn to search for the factors which precipitated the condition.

Aetiology

The aetiology of renal calculi has absorbed medical thinkers for generations. Many factors have come in for their share of accusation or suspicion. The climate was once considered a factor because it was observed that renal calculi was especially common in the tropics. The incidence in India and Egypt was cited because here it was notoriously high.

Then race was to play a part, and it was observed that the negro was singularly exempt from this distressing condition. In like manner sex, habits of living, and even skin texture was investigated but in each case no significant lead was established.

From the maze of theories, folklore and mysticism, with which the whole subject became entangled, two outstanding facts became evident.

1. The growth of a stone is a relatively slow process requiring years.

2. The onset is obscured by its long silence. Some stones may never make their appearance felt at all. Primary calculi have been described as stones having no apparent cause, neither related to urinary abnormalities, obstructions, nor haematuria. But as Randall says, "Fifty percent of cases of renal stone fail to present clinical evidence as to why they occur, after the most careful scrutiny by all the diagnostic means at our disposal." This frank statement indicates the difficulties we can expect to encounter in any individual case when we attempt to answer the patient's honest query:

". . . but doctor, how did the stone get there?"

Modern views, led by Randall of Philadelphia point out four probable conditions which favour nephrolithiasis. The first of these is:

Stasis . . . the stagnation of urine alone is not likely to effect the chances of a stone forming. However, as so often happens the stasis is only one step in the production of an infection. This latter we shall see has a tremendous role. A sterile stasis unless it upsets a colloid balance must not be considered in itself a very important factor.

Infection . . .It used to be thought that a normal kidney would filter bacteria from the blood and excrete them with the urine. This is now known to be untrue, at least in the normal healthy glomerulus. Therefore if today we find a bacilluria we do not examine the blood at once but rather inspect the lower urinary tract.

However, bacteria in the blood or in tissues far removed from the kidney may affect the kidneys by their toxins. It has been shown that the kidneys excrete toxins and at the same time concentrate them. Casman and Mudd have shown this is so with streptococcal toxin. We are not surprised to find epithelial damage in the collecting tubules though the foci are far removed.

Rosenow's experiments are now well known. He recovered streptoccoci from the urine of dogs with renal calculi. After culturing them he implanted them into the teeth of puppies, which had previously been damaged. Later he was able to obtain primary renal calculi from a very high percentage of their renal calyces. It seems probable that in some such manner as suggested above the toxins brought about epithelial changes. The exact processes involved in stone formation are considered later.

One might well ask . . . "even if bacteria were admitted to the pelves of kidneys how could they aid stone formation?" The answer is fourfold:

i. Bacteria in highly concentrated urine disturb the colloidal balance of that urine.

ii. Bacteria in clumps or epithelial debris caused by the bacteria have been proven to act as a nidus of crystallization for urine salts. This might therefore permit a *sterile* primary stone to be formed around a nidus of bacteria!

iii. Bacteria may cause an ulceration of the pelvic wall of a kidney, particularly in the region of the papillae. This we shall see is a most important factor in *Primary stone* formation.

iv. The bacteria may have the property of splitting urea into its components, ammonium carbonate, and this in turn into carbonic acid gas and ammonia. The former causes an alkaline urine which in itself favours certain pathogenic organisms.

Vitamins

Vitamin A deficiency in animals has a very marked effect on renal and urinary epithelium. There is a distinct change in cell type from the cuboidal to the more squamous variety with a subsequent desquamation. However, while in the experimental animals this is certainly a factor it is unlikely to be so in man, whose diet is seldom deficient in this particular substance. Any stones produced experimentally have been observed to have consistently been those of calcium phosphate.

On the other hand Vitamin D is intimately associated with calcium—phosphorous metabolism. It has been asserted that excess of this vitamin may lead to a pathological increase of these substances in the urine. Even if this were the case, however, it should be remembered that hypercalcuria and hyperphosphaturia do not necessitate stones from these substances. As Keyes has remarked in his text, one can have a urine that dries like plaster for years and yet have no evidence of stone formation. He even goes on to say, "Phosphatic stone is never due to phosphaturia."

More likely is it that an imbalance of vitamins, rather than an individual deficiency or excess of any one, is the role played here.

Hyperparathyroidism

The frequency with which renal stones of calcium and phosphorous are found in von Recklinghausen's disease seems to contradict what we have already said about blood levels being a factor in stone formation. The presence of large bony cysts, or fractures resulting from pathological rarefaction are routinely followed by X-ray examination for renal calculi. Blood and urine levels of calcium and phosphorous are greatly elevated, so high indeed that they are precipitated in the urine as salts. Yet the whole story is not inconsistant when we learn of the part played by adventitious calcareous plaques of degeneration in the renal pelves.

Some Modern Aspects of Primary Calculus Formation

Several years ago Randall, realizing the unsatisfactory explanation of stone formation, began an exhaustive investigation into the pelves and calyces of kidneys as they were recovered at autopsy. He made routine thorough examination by hand lenses and microscope of all calyces especially the minor calyx as he realized here was the commonest site of stone formation. He began by studying the normal papillary openings. After many apparently normal specimens had been observed he succeeded in locating occasional small pathological changes. These were

in the form of miliary areas of degeneration with deposition of calcium salts. Some were subepithelial while others were directly exposed to the urine. Then by chance he succeeded in finding a small stone attached to one such plaque. From this he interpreted that he had found the site of formation of this particular stone. By microchemical analysis he found the calcium was often deposited with a substance closely resembling if not identical with nucleic acid. He writes, "We have observed 65 kidney specimens in which calculi have been seen growing upon papillae.

Despite the fact, that the plaque was a calcium deposit, other workers demonstrated by analysis that the stone, attached to the plaque by a pedicle was often of a very different substance. One stone measuring 3 mm. in diameter was found to be a mixture of calcium carbonate and calcium phosphate. In others calcium oxalate, and uric acid stones were found. In these instances he insists there was no evidence under the microscope of an infective process.

Proceeding up the renal tubules he was able to establish other similar plaques in the basement membrane of the tubules, some forming a complete annular plaque around the tubule.

These findings of stone formation are in accord with our present knowledge of calcium plaques and deposits in other sites of degeneration. If this work is corroborated it explains the fact that a stone may grow and remain fixed over a long period of time. As long as it remains attached to its base it is less likely to give evidence of its presence.

If, then, this is true, might we not expect to find evidence of the site of attachment of a primary stone in those cases that are studied early in the stone's descent? In a series of 265 newly voided calculi, 106 visible facets of just such lesions were actually demonstrated. In summing up this work it seems entirely plausible that a calculus is formed on a precalculus lesion which in the majority of cases is some pathological though often minute degeneration in the papillary wall of the minor calyx.

It should be pointed out that this work in no manner excludes the role played by Rosenow's streptococci, their toxins, or metabolic disturbances of calcium and phosphorous. The action of a greatly concentrated toxin may initiate Randall's degenerative plaques. Hypercalcaemia may facilitate deposition of calcium.

The Diagnosis of Renal Calculi

Unfortunately these foreign bodies are rarely suspected or diagnosed until they have given some discomfort or other disturbing symptoms. Only a routine examination would prove the presence of a quiet, though large, calculus. Accordingly we must expect the stones to be well advanced before symptoms are found. These commonly are:

i. *Renal colic,* this extreme pain is caused by preteral muscle grasping the loose stone and attempting to "peristaltise" it into the bladder. If the stone is too large to pass there may be muscle spasm, which causes excruciating agony.

ii. *Haematuria* is commonly observed from ulceration of the urinary epithelium, rupture of ureteral vessels, or degeneration following impaction.

iii. *Evidence of obstruction* in the lower urinary tract.

iv. *Vomiting, shock, cold perspiration* and a *subnormal temperature* with a rapid weak pulse.

X-ray following the retrograde introduction of a radio-opaque substance will show a large stone by filling defects, or without any such substance a large stone may cast its own shadow. Even here the picture may be confused with a calcified mesenteric lymph gland after tuberculosis, a chip fracture of a transverse process of the lumber vertabrae or a concretion in the appendix or even in the gall bladder.

Attempts have been made to palpate the stone "per ureteri" by long flexible metal seekers. The transmission of a gritty sensation to the hand of the operator indicates contact with a stone. Sometimes the blunt point of the seeker is covered with a wax which will retain an impression of the calculus surface.

Course of a Renal Stone

1. The stone, especially if large, may remain in the pelvis of the kidney unknown to its owner only to be discovered at autopsy as an incidental finding.

2. If the stone is smaller it may slip its moorings, be grasped by the upper end of the ureter. This process produces the abovementioned renal colic. If the stone passes the ureter safely it may be voided or cause an obstruction in the bladder or urethra. Growth is still possible to a stone in the bladder.

3. Impaction in the ureter with subsequent ulceration, perforation or hydronephrosis are commonly met in practice.

Higgins of Cleveland in the *Annals of Surgery* for March, 1942, has listed the following five points in the ureter where construction is liable to prevent the passage of small stones. These are:

i. At or just below the junction of the renal pelvis and ureter.

ii. Where the ureter crosses the iliac vessels.

iii. In the female, at the base of the broad ligament, or in the male at the vas deferens.

iv. Where the ureters enter the external muscle of the bladder.

v. At the uretero-vesical orifices.

The ureters between these constrictions tends to spindle out.

4. An impacted stone if untreated may rupture or ulcerate through the ureter and set up a peritoneal-ureteral fistula. If the ulceration is incomplete and healing occurs the constriction is only intensified by the scar tissue.

5. Lastly a renal stone will commonly be associated with inflammation and infection, which is a superimposed condition following obstruction and urinary stasis. In this case the production of a mixed secondary stone from a pure primary one is readily understood.

Treatment

Any treatment of a urinary calculus is purely palliative so long as it fails to rectify the cause of the stone. Removal of a parathyroid adenoma is as imperative as the removal of an offending stone in von Recklinghausen's disease.

But confining ourselves to the calculi alone two methods of approach have been attempted.

A. *The medical approach* has been tried in the hope that a stone will succumb to solution. It was argued that since by solution the salts were deposited by solution they shall be removed. But despite the theory the method has failed to yield results. Lithium salts were once hailed with enthusiasm but have been discarded. The organic matrix which binds the inorganic crystals probably makes the steps of crystallization irreversible. Besides, many chemicals found in stones are relatively insoluble at any possible pH that can be achieved in urine.

B. The surgical approach is adopted when it has been proven that the other kidney is functioning satisfactorily. The exact procedures are beyond the scope of this paper. Suffice it to say that the stone whether in kidney substance or renal pelvis MAY be removed without radical nephrectomy. One operation popular in some centres is the temporary nephrostomy prior to a nephrolithotomy.

If the pelvic stone has slipped into the ureter it may be encouraged to pass to the bladder by forcing fluids, using protective urinary antiseptics and atropine to reduce ureteral spasm.

Ureters are sometimes dilated with bougies to facilitate passage of a proven small stone.

Finally when other methods have failed and obstruction is evident operation must be resorted to. This latter procedure can be performed extraperitoneally and with remarkable low mortality.

Summary

Renal calculi have been reviewed from the standpoint of classification, aetiology, diagnosis and treatment. The most prominent factors in their aetiology have been reduced to stasis, infection, vitamin imbalance and hyperparathyroisism. The part played by plaques of calcium salt in the renal papillae of the minor calyces have been investigated. The evidence at hand points to a close correlation between a degenerative process at this site to primary renal stones. The diagnosis of nephrolithiasis is largely clinical and radiological. The treatment is for the most part straightforward and the results are excellent. It has been emphasized that any urinary calculus, no matter what its urgency, is always a secondary condition following on a precalculus lesion or pathological state.

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HEALING OF FRACTURES

H. B. STEWART, '45

In reading the literature prior to writing this article on what had originally appeared a relatively limited subject I have found that there is a very extensive discussion of the problem in a great many aspects. In such a short article it is impossible to summarize even small parts of the published data so I have purposely included a great many references which should give anyone interested an introduction in any of the many branches of the field.

Healing of fractures on superficial classification lends itself well to discussion under two headings:

(A) Chemical changes in fracture healing.

(B) Structural changes in fracture healing.

Since the clinical approach to fractures is beset with opinions and experiences and all too little controlled observation it seems desirable to discuss the chemical basis first and then try to correlate the multitude of clinical observations.

PART I

Calcium Metabolism with Special regard to fracture repair. These may be further discussed under certain headings:

(1) Maintenance of Calcium Balance in the body:
  (a) Absorption
  (b) Excretion
  (c) Parathyroid hormone.

(2) Blood Calcium.

(3) Precipitation of Bone Salt:
  (a) Solubility Product
  (b) Changes in the nature of $Ca_3(PO_4)_2$ precipitate
  (c) Nature of Bone salt
  (d) Logan's theory of bone salt deposition.

(4) Method of Calcification.

(5) Special Controls of Calcium metabolism Hormonal and Vitamin.

1. *Maintenance of Calcium Balance*

(a) Absorption of calcium. This comes from the food in the form of inorganic phosphates, glycerophosphates and bound in the structure of proteins (e.g. casein). Absorption is favoured by the following factors:

(1) Acid forming substances in the diet which keep the bowel at an acid pH favour absorption. These include lactose (1), oils (2) and buffer mixtures such as citric acid-sodium citrate mixtures (3).

(2) Vitamin D specifically activates the absorption of calcium from the bowel according to Nicolaysen and some American studies (4).

(3) Depleted calcium stores increase the uptake of calcium while large stores on the other hand depress it (5).

(4) Parathyroid hormone has no function in the absorption of calcium (6).

Certain other factors tend to inhibit or limit calcium absorption:

(1) Alkalinity of the bowel in the region of absorption.

(2) The presence of oxalate in the bowel even in the presence of Vitamin D limits absorption (7).

(3) Cereal diets which by analysis have been shown to contain adequate cal-

cium do not liberate from the unutilizable phytin-form absorbable calcium (8).

(4) Chronic catarrh of the small bowel and the formation of calcium soaps in steatorrhea both limit the calcium absorption (9), (10).

(b) Excretion of calcium has long been attributed to the large bowel as a result of the calcium containing stools in hyperthyroidism. Evidence of increasing calcium content of fecoliths descending the large bowel has also been presented. However, in this connection it is well to note that calcium given intravenously is excreted almost quantitatively by the kidney. Also increased Vitamin D administration decreases the fecal calcium and increases the urinary calcium (4). Fecal calcium therefore as stated by Logan is not an active excretion process but rather incidental with increased intestinal secretion (11).

(c) Parathyroid hormone. The primary effect of parathyroid hormone is to increase the urinary phosphate and cause a fall in the blood phosphate (12). Since the $Ca \times PO_4$ product in blood is constant, a rise in calcium follows the fall in phosphate, the source being bone. Now the kidney is adjusted to handle a certain concentration of calcium and when this is exceded the calcium overflows in the urine. This tentative explanation would suggest that parathormone acts primarily by altering the kidney threshold to phosphate (13). This is supported by the fact that parathormone injection in animals with a bilateral nephrectomy causes no rise in the blood calcium (14).

Later experiments on similarly treated animals, however, showed that bone resorption did occur and was presumably excreted elsewhere (47).

Work done by Ham indicates, however, that parathormone may not act in this manner. He finds that hypercalcaemia produced by parathormone injection far exceeds that which can result from simple phosphate excretion and he suggests the alternative theory that parathormone acts by formation of a complex between the calcium and the hormone which slowly breaks down as the effect wears off and this breaking down complex so increases the concentration of calcium that pathological calcifications occur in the aorta, coronary arteries, kidney, etc. (44), (45).

It is to be noted pathologically, that hyperplasia of the parathyroid in recorded in chronic renal diseases particularly with the contracted type of kidney. Work done by Ham and Tisdall show that such a hypertrophy of the parathyroid can be produced in animals *only* by low blood calcium and not by low phosphate (48).

2. *Blood Calcium*

This matter has been the subject of exhausting researches. The general conception is that calcium exists in 2 forms as outlined in table.

(A) Indiffusible calcium
40-55% 4 –5.5 mgm/cent
(1) bound to protein
(2) colloidal calcium phosphate

(B) Diffusible calcium
45-60% 4.5 –6 mgm/cent
(1) ionised calcium
(2) unionised diffusible form
Total.....10 mgm/cent

A. (1) That portion bound to the protein appears to be in the form of calcium proteinate the dissociation constant of which compound is $10^{-2.22}$. The relation of the total calcium to the bound calcium for normal individuals is expressed as a straight line equation

Total calcium $= m$ (protein calcium) $+ b$

$$[Ca_T = m\ Ca_{P_r} + b]$$

where m is the slope of the line and b is the y intercept. Separation of this equation can be accomplished into 2 portions which are referable to the albumen and globulin fractions of the blood and as would be expected the greater portion is bound to the albumen. The calcium content of protein is directly proportional to base binding properties varying as pH and this suggests that no internal linkages occur.

(2) The colloidal calcium is denied to be normally present by some authors but claimed to be present by others. Its function is unknown (15), (16) and (17).

B. (1) The ionised calcium is determined by the method of McLean and Hastings (18) using frog heart systole as the criterion. They, and later Logan, found that solubility product as it is found in blood was $10^{-23.1}$ (19).

(2) The unionised diffusible calcium appears to be in the form of a calcium citrate-like compound and constitutes 5% of the total calcium.

3. *Precipitation of bone salt*

(a) Solubility product. Our present knowledge of the mode of precipitation of bone salt is largely attributable to the work of Logan and Taylor (20), (21), (22), (23), (24).

It is known that calcium phosphate precipitates when the product of the calcium ions cubed and the phosphate ions squared exceeds a constant termed the solubility product. For a long time the solubility product of this compound was thought to be 10^{-27}. It was also found that the solubility product determined in fluid in equilibrium with bone salt was 10^{-27}. The principal objection to this seemingly compatible view is that the solubility product of these ions in blood is 10^{-23} as mentioned previously. That is the blood was 200% supersaturated with respect to bone salt.

(b) Changes in a Calcium phosphate precipitate. Logan's principal contribution to this problem was that he demonstrated that the solubility product, by definition a constant, varied depending on the amount of solid equilibrated with the aqueous phase in the process of the determination. He found that it varied from about 10^{-23} with one milligram of precipitate per litre to about 10^{-27} when a great excess of precipitated phosphate is present (100 mgm/litre). This led to the propounding of a theory that calcium ions were adsorbed into a precipitate after it was formed consequently altering the concentration of the calcium ion in the equilibrated solution and thereby altering the solubility product. The amount of change is proportional to the amount of precipitate present. He also demonstrated that phosphate was eliminated from the precipitate after it was formed and that if carbonate ions were in the equilibrium fluid even though their concentration be less than the solubility product of $CaCO_3$, they were none-the-less removed from the solution and taken up by the precipitate. The process is at first rapid but later slows down as saturation is approached and can be measured by serial analysis. If no carbonate ions were present, the elimination of phosphate still took place and the replacing ion was presumably hydroxyl.

(c) Nature of the bone salt. Logan demonstrated with corroboration from other sources, that bone salt as precipitated had the empirical formula of

$[Ca_3(PO_4)_2]_n.CaCO_3$ (*n* is 3 in children and 2 in adults). $MgCO_3$ may be substituted in place of carbonate and fluorides may replace carbonate ions. In the formation of such a compound there are 2 possibilities. Either it precipitates as one large aggregate or in stages. The geological counterpart of this compound, carbonate apatite, is considered a true chemical entity precipitating in one mass. It has a known X-ray spectograph which is identical with $Ca_3(PO_4)_2$. Logan does not believe that bone precipitates in this manner but rather forms calcium phosphate which adsorbs calcium carbonate. Adsorbed ions do not alter the X-ray spectograph and there is no evidence of $CaCO_3$ spectograph in the X-ray examination of bone.

(d) Logan's theory of bone salt deposition. The initial stage is the precipitation of $CaHPO_4$ for he finds that calcium and phosphate precipitate primarily in a ratio of 1:1. This is rapidly altered by the formation of ionic complexes which involve 3 mols of $CaHPO_4$ from which is eliminated one mol of phosphoric acid leaving therefore tricalcium phosphate in a crystalline lattice form which can be identified by X-ray diffraction. $CaHPO_4$ crystals cannot be demonstrated either by microscope or X-ray at the time, at which the determinations were necessarily made. They are probably present in earliest stages. The tricalcium phosphate then having been formed proceeds to adsorb carbonate ions and with them calcium ions leaving the final solubility product of 10^{-27} and a compound with the formula of

$$[Ca_3(PO_4)_2]_n.CaCO_3.$$

Evidence for adsorption is adduced by treating bone salt and in vitro precipitates with hydrochloric acid. It is found that 47-56% of the carbonate can be removed with the removal of only 6-9% of the phosphate thereby suggesting that carbonate lies on the surface of the crystal between the layers of the phosphate molecules.

4. *Method of Calcification*

If the previous thesis is accepted as correct, precipitation does not take place out of supersaturated solution. In fact the solubility product of $CaHPO_4$ is $10^{-5.6}$ which is immensely greater than the blood level of $10^{-23.1}$. (Incidentally, it is to be noted that measurement methods are sufficiently inaccurate that it is uncertain whether the blood is saturated, *slightly* supersaturated or *just under* saturation with respect to $Ca_3(PO_4)_2$).

Such a high concentration of Calcium as is necessary to give this product ($10^{-5.6}$) is obviously the result of a vital process for simple blood changes would be insufficient to produce such a change. Enzyme systems seem to be the important factor. Note the following facts.

(1) Phosphatase occurs in cartilage just preliminary to ossification (when the cells are lying in dilated lacunae) and cannot be isolated from non-ossifying cartilage (25), (26).

(2) Phosphatase causes calcification in vitro in presence of glycerophosphate but not in the presence of an equivalent concentration of inorganic phosphate. Higher concentrations of inorganic phosphate will result in calcification.

(3) Treating bones with fat solvents, cyanide, fluoride, iodoacetate, etc., which do not paralyze phosphatase prevents calcification from *inorganic* salts.

This and other evidence leads to the conclusions that phosphatase is undoubtedly concerned in calcification, and,

that another system, either a distinct enzyme system, or a substrate preparing system is necessary for the handling of the inorganic phosphorous. The action of phosphatase is presumably to increase PO_4 concentration and thereby exceed the solubility product for $CaHPO_4$.

Tollman and others have shown that phosphatase in callus of healing fracture varies directly as the oxygen consumption of the tissue (28). The same group of workers showed that in infected fractures no healing appeared until phosphatase appeared and that phosphatase is markedly lower in the septic fracture than in asceptic fracture, as long as the infection persists (29).

Thus calcification seems to represent the combination of the vital and non-vital factors in bone change and unless both are intact no bone deposition or resorption can occur.

5. *Special Control of Bone Metabolism*

A. Hormonal.

(1) Parathyroid hormone. This has been discussed as a method for the gross movement of calcium and is probably not greatly concerned with fractures except in the maintenance of blood calcium. It has also been shown that it activates phosphatase (13) and decreases blood phosphatase (30) and acts through formation of complex.

(2) Thyroid. Hyperthyroidism causes a negative calcium balance (31) though healing of fractures (32). It may, however, retard the rate of growth of bones during the growth of bones during the growth period (33).

(3) Sex hormones. There is a gradual fall in the blood calcium during pregnancy according to Myer Bodansky. It has also been noted that calcium is permeable to the placenta *only* in the direction of the fetus (34).

(4) Other hormones adrenal, follicular, thymic, etc., are known to affect bone deposition.

B. Vitamin Effects.

Vitamin D is known to act by increasing absorption as mentioned. Certain effects on bone cells have been noted. (1) Vitamin D in vitro causes calcification of the growing area of the tibia (35). (2) Vitamin D increases the deposition of phosphate in the skeleton of some animals by 25-50% (37). Action may be through phosphatase.

Vitamin C. This is important in the repair of fibrous stroma of bone. Vitamin effects are more completely discussed with regard to the structural changes recorded, for the experimental work uses histological criteria for evidence of change. These will be included in the second portion of the paper.

PART II

Histological changes at the fracture surfaces

There are a series of changes which have been variously described by various authors. The most extensive discussion of the subject and the best evidence that has come to my attention is presented in a symposium published by Hertz (37) in which he describes a series of experiments on the healing of fractures with special regard to the diet and its vitamin content. As such, his experiments represent the best-controlled series of experiments available in the literature and this portion of the article is devoted to a summary of his findings with notations on his terminology and the views of certain other authors.

The discussion is best divided into parts according to the serial sequence of the repair as follows:

1. Hemorrhage.
2. Primary Apposition of the fragments:
3. Secondary Apposition of the fragments.
   A. (a) Direct Ossification
      (b) Indirect or Endochondral Ossification.
   B. Calcification.
   C. General Changes in the Bone.
4. The precise function of the vitamins in repair.
   (1) Vitamin C.
   (2) Vitamins A and A and D combined.
   (3) Effect of Rickets.
   (4) Hypervitaminosis-D.
   (5) Other Changes referable to vitamins.

1. *Hemorrhage.* According to Leriche and Policard (38) whose findings were based on gross observation largely the hemorrhage that occurs coincident with the fracture is organized into the Callus. This view is not now generally entertained and Hertz states that in a "normal" fracture all the hemorrhage has been removed by the 4th day. (His fractures were made under uniform conditions and by a uniform method.)

He also states that resolution of the hemorrhage is retarded in the scorbutic animals and to a lesser extent in A and A-D avitaminoses. Hemorrhage is accompanied by cellular infiltration to an extent proportional to the trauma to the soft tissues.

2. *Primary Apposition of Fragments* (presuming manipulation into position). Healing according to Hertz commences as an inflammatory proliferation of the cambium layer of the periostium and to a lesser extent the endostium and by the cells of the extreme ends of the marrow cavity. Scorbutic animals show a noticeable delay over normal animals in the formation of this traumatic inflammatory response and it does not appear until the 8-10th day and then is scanty. Previous to this time the fragments are passive. Thus in all animals primary union of a fracture is by osteogenic granulation which is markedly vascular except in hypervitaminosis-D. The granulation so formed shows an abundance of collagen which is largely absent in a C-avitaminosis.

3. *Secondary Apposition of the Fragments.* This can occur by direct ossification or by an intermediate stage of cartilage formation. Both types of healing apparently occur in the body. In the opinion of Thomson and Miles (39) cartilage formation is favoured (1) by incomplete immobilization and (2) in bones which are embryologically endochondral. In the opinion of Ham cartilage formation is the result of avascularity alone.

A. (a) Direct Ossification: (Based on observations recorded by Hertz). The following represent the cellular changes in direct ossification.

The proliferated cambium layer of the periostium which has anchored in primary union the two fragments undergoes a change which is evident the peripheral part of the cytoplasm as opposed to the perinuclear cytoplasm. Collagen fibrils are laid down in bundles and bind the fragments together. Similarly they bind the individual cells to each other in the manner of a syncytium which involves the peripheral cytoplasm only. Into this syncytium calcium salts are deposited

giving rise to true bone. Since Vitamin C inhibits the formation of collagen, it is apparent that this process is prevented in scorbutic animals. B_1-B_2, A and A-D avitaminoses do not alter this process.

The potentialities of the cells do not seem to be altered by the collagen deposition for on the surface of this callus free proliferation can take place with the formation of additional syncytium and eventually bone.

(b) Indirect Ossification: This process differs from the process just described in that there is an intermediate stage of cartilage formation. Hertz describes the formation of "chondroid" in the peripheral cytoplasm just as the collagen was laid down which becomes altered into "chondrin" which equivalent to our basophilic cartilage (the chemical nature of these compounds is obscure). Thus the primary callus becomes converted into cartilaginous callus (callus denoting the swelling at the site of union of the fragments). Scorbutic animals and Vitamin A free animals will not form cartilage. B_1-B_2 avitaminoses and D hypervitaminosis do not affect the process while rachitic animals (from the McCollum diet 3143) show redundant cartilage formation.

The alteration of this cartilaginous callus to bone represents the second stage in indirect ossification and occurs in a manner comparable to that described for the ossification of cartilage in the fetus. Degeneration of the cartilage is seen with the formation of calcium desposits in the matrix (it might also be noted here that Shohl has demonstrated that calcium is present in much greater quantities in cartilage protein than in serum protein). The cells undergo hydropic change and similtaneously there is invasion of the cartilage by highly vascular buds of the original osteogenic proliferation. These dissolve the cartilage before them leaving incidentally a high concentration of calcium in solution. When the process is complete it leaves according to Hertz undifferentiated connective tissue which is free to proceed to direct ossification. Thus from a teleological point of view the function of cartilage is to behave as a splint until the body is mobilised to repair the fracture. From a functional standpoint it is a simple result of avascularity which is corrected at the time of dissolution of the cartilage.

In the rachitic animal the cartilage changes are not the same but are believed to be a metaplasia of the cartilage into osteoid tissue which is not regarded as true bone.

The essential point in ossification seems to be vascularization (Ham, 27) and Lexer (40) reports the failure of union presumably due to calcium deposition in the vessels preventing adequate "fracture hyperaemia".

B. Calcification. The method of calcification has been described in part I. Certain special cases remain to be mentioned. (1) The scorbutic animal shows excessive calcification to the extent of brittleness. (2) B_1-B_2, A and A-D avitaminoses did not impede the appearance of calcification centres of the callus. (3) Rachitic animals on the other hand showed no calcification.

The appearance of trabeculae is always remote from the fracture line first. Robinson believes this is due to the immobility at the site and it is not influenced by dietary changes.

C. General Changes in Bone. A fractured bone undergoes a decalcification evidenced by a less dense X-ray shadow.

Two factors are at work here. Absorption of necrotic bone resulting from fracture shock and ischemia and absorption of viable bone with an increased diameter of the Haversian canals. Hertz introduces an interesting point of view here. Since he believes that all the bone skeleton aside from the calcium salts is part of the exoplasm (matrix) or endoplasm (as is seen in the lacunae) of the bone cells, therefore, when the separating calcium salts are removed, these elements coalesce into a giant cell or an osteoclast and in this resorption process he describes the existence of intracortical osteoclasts. Ham has also recorded similar findings though in his opinion most osteoclasts do not arise in this manner.

In this manner large parts of the bone may be removed and replaced by ingrowing osteogenic cells. This is limited in the scorbutic animal while in the rachitic animal the absorbed bone is replaced by osteogenic tissue which does not become calcified.

4. *The Precise function of the vitamins in repair*

(1) The scorbutic animal may show no callus until about the 26th day and it is quite incapable even after this time of holding the fragments together. This is due to (1) a decreased formation of osteogenic tissue, (2) defective collagen content, (3) excessive calcification to the stage of brittleness, and (4) cartilaginous foci are absent. In addition to this, the ends of the bone shrink away from each other due to the disappearance of the necrotic part without osteogenic replacement.

(2) A and A-D avitaminoses while preventing the formation of cartilage focus do not prolong fracture healing or produce inferior union.

(3) In rachitic animals the failure in union is not the failure in production of osseous tissue but a failure of this tissue to become calcified.

(4) D-Hypervitaminosis seems to favour rapid healing by direct ossification. It gives complete firmness at the site of fracture early but it is accompanied by fragility and brittleness. As true ossification proceeds following a cartilage calcification in indirect union, the bone formed is equally as good as the original bone and is formed much more rapidly than the untreated fracture. Massive doses of Vitamin D therefore evidently stimulate fracture healing.

(5) Pure D-avitaminosis in the presence of adequate salt does not apparently hinder the healing of fractures though this seems inconclusive. Phosphate deficient diets do hinder fracture repair (41), (42), (43). Prolonged rickets or prolonged scurvy seem to preclude proper healing of fractures in spite of adequate intakes. These latter points may be factors in the production of pseudoarthroses.

Summary

The author has tried to present in review the important aspects of fracture repair. These were not discussed from a clinical point of view with respect to treatment but from a chemical and histological approach in an effort to emphasize the aspects of diet and the pure mechanics of the deposition in order that treatment may be directed to satisfying these demands.

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THE COMPARATIVE ANATOMY OF THE FOOT AND ITS RELATIONSHIP TO FUNCTIONAL DISTURBANCES

W. R. HARRIS, '46

Although the human foot is a more specialized organ than the human hand, it departs but little from the basic reptilian pattern. This fact is true in all primates. In order that this type of foot may support weight, it presents many interesting anomalies, found in the skeletal, ligamentous and muscular make up of the foot. The scope of this article will be limited to a discussion of some of the skeletal defects. Before the nature of these defects may be properly understood, it is necessary to have some knowledge of both the comparative anatomy and physiology of the region.

Comparative Anatomy

The development of the human foot runs, in many respects, a parallel course to the development of the foot in the apes, but although anomalies of the foot in man may sometimes present conditions very similar to the normal foot of say the orang outang, it cannot be said that these anomalies are a result of direct inheritance from that animal.

The foot of the anthropoid ape marks the climax of a long series of developmental changes. These changes began with the reduction of the many-rayed fin of the fish to the basic five-toed reptilian pattern. This foot possessed movable joints, and projected from the body, thus lifting the animal from the ground. Such a pentadactylic foot possessed nine tarsal bones, and is represented by the foot of the present day turtle.

Primates, walking erect, throw the whole weight of the body on to the lower limb. This phenomenon produces changes in the foot that are characterized by:

(a) An enlargement of the weight bearing tarsals, namely the talus and the calcaneum.

(b) Disappearance of the os intermedium by fusion with the talus (os trigonum represents an ununited os intermedium).

(c) Progressive "climbing up" of the talus from its position medial to the calcaneum to a position on top of the calcaneum. This movement has been described as a rotation of the talus on the calcaneum.

Furthermore, the apes are adapted to an arboreal existence, and hence their feet are more specialized than human feet. This is evidenced by opposition of the hallux to the remaining digits. The degree of opposition is, I think, directly related to the amount of rotation of the talus on the calcaneum, i.e., in a foot in which the talus has not climbed very high up on the calcaneum, there is a divergence of the bone directly attached to the head of the talus, namely the navicular bone. This "falling down" of the navicular is probably the cause of the divergence of the first metatarsal and phalanx. This adaption to arboreal existence is further marked in the apes by an inversion or more correctly flexion of the remaining digits. Thus the ape when walking supports its weight on the outer side of the foot.

The foot of man marks the climax of a separate branch of development, for only in the human is there enough eversion of the foot to allow the entire sole to rest

on the ground, and only in the human foot has the talus mounted so completely on top of the calcaneum. Loss of opposition of the hallux is a further characteristic of the human foot. In certain conditions the foot presents anomalies that strongly resemble the foot of the apes, namely a looseness or hypermotility of the first metatarsal segment which involves opposition of the great toe, and is frequently associated with a lowered head of the talus. This condition will be described in detail later.

Physiology of the Foot

(a) *The foot in stance*

In man, when standing erect, the center of gravity of the body is located in the mid-line just in front of the promontory of the sacrum. From this position the line of body weight passes perpendicularly through the center of the head of the femur, through the front of the knee joint, and as far in front of the ankle joint as the fore part of the navicular bone. Thus there is a tendency for the body to fall forwards and this is prevented by a continuous slight contraction of the powerful muscles on the back of the thigh. Erect stance, then, involves a minimal muscular effort.

When discussing the foot in stance it is well to remember that the normal individual has two feet, the position of both of which must be taken into consideration. There is a considerable variance of opinion as to the proper angle between the feet, but the consensus of opinion seems to be that an angle of thirty to forty degrees is necessary to give the body proper lateral support (Morton). In this position the line of the center of gravity passes between the feet in the plane of the scaphoid bones. The head of the fifth metatarsal bone marks the limit of lateral stability. Anteriorly the limit of support is marked by a line joining the heads of the first metatarsals, while posteriorly it is a line joining the medial calcaneal tubercles. Any shift of body weight that causes the line of gravity to move out of the area just outlined upsets the equilibrium and is accompanied by increased muscular tension to counteract it. For example, forward bending of the trunk causes the line of force to pass forwards out of the central area of support and this is compensated for by an instinctive contraction of the long flexors of the toes tending to extend the ankle joint and thus restore the normal equilibrium.

The effectiveness of the foot in supporting body weight is dependent not only on its bony trabecular structure but also on the cohesion afforded to its units by the ligamentous tissues. The weight of the body standing erect passes equally down each leg and is transmitted to the foot. The antero-posterior arching of the foot then flattens out until the plantar ligaments are taut and hold the whole in a solid arch. The weight is then distributed to the points in contact with the ground via the structure of the bony trabeculae of the individual bones of the foot and the ligaments of the plantar surface.

By means of a piece of apparatus consisting of two sets of scales each of which consisted of three weight bearing plates connected to separate scales, Morton has determined that the normal weight distribution in a 120 pound man is as follows:

To each foot	60 lbs.
To heel (medial calcaneal tubercle)	30 lbs.
To head of metatarsal I	10 lbs.
To head of metatarsals II to V	5 lbs. each

The weight is thus evenly distributed between the anterior and posterior halves of the foot. Because the head of the first metatarsal bears twice the weight of each of the remaining metatarsals, it has two bearing points, the sesamoid bones.

The above data must not be accepted as conclusive, as the "normal position" of the body in stance depends not only on the opinion of the individual investigator, but also on the physical condition of the subject. Obviously the picture will be changed in an obese individual.

These results show that the two medial metatarsals bear as much weight as the three lateral metatarsals. Thus an "axis of balance" is described in the foot as being the plane passing between the heads of the second and third metatarsals. This is to be distinguished from the axis of leverage, through which weight is transmitted when one stands on the toes as in walking and running.

It has been shown that the normal foot will maintain its shape even under deep anaesthesia, indicating that its shape is primarily due to its ligamentous and bony configuration. Due to their structure, however, ligaments are adopted to withstand sudden stresses and strains, not prolonged usage. Hence any defect of the foot shifting the axis of balance out of its normal position causes undue strain on the ligaments, with subsequent stretching, and spreading of the tarsal bones.

(b) *The dynamic foot*

Weight distribution in the moving foot presents a very different picture than the foot in stance. Moreover, the structure of the foot conforms to functional necessities.

During walking, the first part of the foot to strike the ground is the medial calcaneal tubercle. As the sole of the foot is placed on the ground the weight is supported on the lateral edge of the foot and the head of the fifth metatarsal. The last part of the foot to reach the ground is the head of the first metatarsal. Thus the "walking" surface of the foot is described as a tripod. This enables rapid adjustment to irregularities of the ground during walking.

The muscles of the foot are concentrated along the medial side, because this is the last part of the foot to reach the ground, and hence it takes the onus of shifting the weight on to the toes preparatory to the next pace. The muscles to the hallux are enlarged, as are also the bones of the first metatarsal segment. As the lateral side of the foot is concerned with weight bearing, and not weight shifting, it has increased ligamentous development in the shape of the long and short plantar ligaments.

During walking, the body weight is shuttled from side to side, from the head of the first metatarsal of one foot to the heel and head of the fifth metatarsal of the other foot. As the line of gravity of the body weight is still a perpendicular from the promontory of the sacrum, each foot must be placed as nearly *under* the body as possible. Thus the feet strike the ground approximately along a single straight line. This is particularly noticeable in running. The toes must be pointed forward, for if they are diverged laterally, the medial side of the foot strikes the ground first and there is a tendency toward fallen arches.

The shape and balance of the normal human foot are dependent entirely on the close interrelation of its muscular, ligamentous and bony structure, and hence it should be pointed out that although the defects about to be described are of the skeletal structures of the foot, they pro-

duce changes in the musculature of the foot, and conversely changes in muscular tension can equally well produce skeletal deformities. Moreover, it is often hard to state that a foot defect is entirely due to either muscular or to skeletal defects. This point will be seen later.

Anomalies

Hypermotility of the First Metatarsal Segment

Lax ligaments on the plantar surface of the foot, notably the inferior calcaneonavicular ligament, allow the first metatarsal segment (metatarsal I and medial cuneiform) a greatly increased range of dorsiflexion. One of the effects, therefore, is that when the foot is placed on the ground, the head of the first metatarsal moves dorsalwards off the ground, and the weight normally born by it is taken over by the head of the second metatarsal. As the head of the first metatarsal normally bears a double load, the load on the second metatarsal is consequently tripled. In an effort to bring the first metatarsal in contact with the ground, the foot is everted. The laxity of the ligaments (hypermotility) will determine the amount of eversion. In bad cases the foot will be everted until the inverting muscles (tibialis posterior and flexor digitorum longus) prevent any further movement, or until the first metatarsal is in contact with the ground. A very similar condition is produced by paralysis of these same muscles. The result is an exaggerated lateral divergence of the toes, with an upward rolling of the lateral border of the foot. Changed muscular and ligamentous strains throw the tarsal bones out of even contact with one another. The sustentaculum tali increases its obliquity in order to lower the head of the talus, with a consequent inward rotation of the leg. In extreme cases as described above, the foot becomes flattened and loses its function as a lever and the person is reduced to cumbersome "heel walking".

Short First Metatarsal

The axis of leverage as described in a preceding paragraph normally passes between the heads of metatarsals I and II, because they are equidistant from the medial calcaneal tubercle. Hence they share the weight in walking and in running. If one of these metatarsals is longer than the other, a disproportionate strain is thrown upon it, and foot deformities result.

A short first metatarsal is a fairly common congenital foot defect. As explained, the weight of the foot is shifted to the longer metatarsal, in this case the second. This results in a large callous developing on the sole of the foot beneath the head of the second metatarsal, with increased growth of the ligaments around the second metatarsal and middle cuneiform. The result is a very painful foot, and often there in inflammation of the tendon sheath of the second toe.

In extreme cases, this defect may prevent the head of the first metatarsal from coming in contact with the ground, and therefore cause the disturbances of function described under *Hypermotility of the first segment.* The condition is par-particularly exaggerated in women who were high heeled shoes.

Metatarsus Primus Varus

This is a deformity wherein the first metatarsal is displaced medially, so that the bony structure of the foot closely resembles the grasping foot with the opposed hallux of the primates. It is frequently found in company with hypermotility of the first metatarsal segment,

and it produces the same changes in the foot, resulting in weak longitudinal arches.

Hallux Valgus or Splayed Toe

Hallux valgus is a deformity frequently a result of metatarsus primus varus, although it may also be traumatic. The heavy eversion of the foot, as described above, causes the toes to be splayed laterally. When the first metatarsal diverges from the rest of the foot, a considerable angle occurs at the metatarsal-phalangeal joint as the hallux makes an effort to regain its normal position. This causes a prominent projection or bunion on the medial border of the foot. This condition is of course exaggerated by wearing short, pointed-toe shoes. Its presence increases the weakness of the longitudinal arch of the foot.

Conclusion

In an investigation now being conducted by H. A. Cates of the Dept. of Anatomy, some curious relationships between hypermotility, short first metatarsal and the degree of rotation of the talus on the calcaneum have been brought out. Of several specimens examined because they had short first metatarsals, nearly all had only a small amount of rotation of the talus on the calcaneum, and furthermore, the talus had slid forward on the calcaneum, apparently in an effort to move the head of the first metatarsal forward as far as the second. Many other specimens had varying degrees of rotation of the talus on the calcaneum. It is more than likely that this is the cause, not the result, of hypermotility of the first metatarsal segment. It is just possible, therefore, that lack of the proper degree of rotation of the talus may be the cause of many foot defects, but that the actual condition is masked by the predominant symptoms of flat fet.

The author wishes to express his gratitude to Dr. H. A. Cates for the help and advice given him in the preparation of this article.

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A HISTORICAL OUTLINE OF THE PROGRESS OF THORACIC SURGERY

R. C. LAIRD, B.A., M.D., M.S., F.R.C.S.(ENG.)
FELLOW IN SURGERY

(From the Department of Surgery, Toronto Western Hospital)

In any full discussion of the problems of surgery of the thorax, one should include diseases of the heart, lungs, pleura, pericardium, oesophagus and chest wall that have a possible or well-established surgical method of treatment. Due to the frequency of such diseases in the lungs and pleura, and the relative infrequency of conditions in heart, pericardium or oesophagus that can at present be relieved by surgical means, this specialty of thoracic surgery may generally be considered to be the surgery of the lungs, pleura and chest wall. The application of surgical treatment to diseases of the heart and pericardium is definitely established, but it might still be considered to be in its infancy, and to be compared with the surgery of the lung of fifteen years ago. Rapid advances have been made in these fifteen years, and we may look forward to similar improvements in cardiac surgery in the near future. The problems in chest surgery have been in diagnosis, in the recognition of the physics of the thorax, and the effect of disturbance of respiratory balance, in the surgical shock attendant on major procedures in the thorax, and in the experimental and practical improvement of various technical operations.

There can be little doubt that the great progress in thoracic surgery in the past fifty years was instituted by a marked improvement in methods of examination. Not only did the ordinary methods of percussion and auscultation and the interpretation of findings improve but there were added such specific methods as X-ray, bronchoscopy, and bronchography. These aids to diagnosis greatly improved the recognition and treatment of diseases already familiar to the surgeon, and opened up vast new fields which had undoubtedly been explored by the most daring, but were now to become the sites of almost everyday operative procedures. Of these various methods, the product of Roentgen is the most outstanding. The radiological progress of any thoracic disease is outlined and followed with interest, and this has been increased again in recent years by the use of the laminograph or planograph, which is particularly useful in the identification of cavities in lung.

This historical outline will deal chiefly with the progress of surgical methods of treatment of disease of the lungs and pleura. The improvement in our knowledge of the physiology of the chest, and the application of this to anesthesia have been two very important factors in this progress, and positive pressure anesthesia may be considered an absolute essential in major chest surgery. The diseases have been divided into suppurative conditions of lung and pleura, pulmonary tuberculosis, and neoplasms, and discussion includes the earliest attempts and what may be considered the established modern treatment.

Suppurative Conditions of Lung and Pleura

Historically, the first chest condition treated surgically was undoubtely em-

pyema. Records show that Hippocrates favoured free incision and drainage of an empyema cavity, and it is likely that there were various methods of treatment used before his day. Later this became unpopular, and there was a swing to the more cautious attitude of medical treatment only for empyema. This period of conservatism was a long one, persisting until Lister introduced antisepsis, at which time many old operations were tried again. In spite of the antiseptics, the wide-open drainage of empyema was still attended by a high mortality and the reason was not definitely established for some years. About the time of Lister, Bülau introduced continuous intercostal aspiration and was definitely on the right track, but this method had few adherents until after the influenza epidemic of 1918. The mortality from acute empyema in this epidemic was shockingly high, because rib-resection and open drainage was the established surgical treatment, and the resultant open pneumothorax gave the patient sufficient added respiratory embarrassment to produce a fatal result. Since that time repeated aspiration followed by closed intercostal drainage saved many patients in the acute stages, and later loculation of the pus could safely be drained by the open method. This general procedure is now recognized by most as being satisfactory, with the inevitable modifications. It is still not possible to draw conclusions as to the effect of sulphonamides on empyema, but there is no doubt that the incidence of this complication of pneumonia is considerably decreased since the use of these drugs. Once suppuration has occurred, the drugs seem to be less effective.

The surgical treatment of lung abscess was for many years a method of last resort. It was definitely established toward the end of the last century by such men as Garré, and Tuffier, and Sauerbruch, who planned external drainage deliberately before it was too late. There was very great difficulty in recognition and localization, however, and most abscesses were drained very late and there was a high mortality. In the past fifty years the first important advance was in localization, which was made possible by the X-ray, and the two-stage drainage became a recognized procedure. The next step did not come until the publication by Neuhof in 1937 of his results using the early one-stage drainage. He stressed the importance of very accurate localization, and of very early drainage. He claimed that the lung was always adherent to the parietal pleura over the abscess, and early drainage was safe if localization was corect. His reported mortality figures were almost unbelievable when compared with previous statistics, but his methods have been used with equal success by others, and the whole outlook for lung abscess has been made immeasurably brighter.

Bronchiectasis was recognized as a clinical entity some years before the beginning of this century. There is an excellent description of this disease in the 1909 edition of *Osler's System of Medicine*. Practically the only important features that have been added to our knowledge of the disease since then have been the outlining of the lung by lipiodol, and the surgical treatment. As collapse therapy became recognized as of great benefit in pulmonary tuberculosis during the first two decades of the twentieth century, the various methods were used in the treatment of bronchiectasis. Generally speaking, they have all been discarded as being of very little use, and some of definite danger. In localized bronchiectasis the logical treatment is removal of

the diseased lung and the operation of lobectomy (first done successfully by Körte in 1907) was introduced into modern surgery by Brunn in 1929. In the same year Toronto is proud to have on record the first lobectomy done by Dr. Shenstone and Dr. Janes, who introduced the snare type of lung turniquet. Their technique was rapidly adopted by many thoracic surgeons, and the number of lobectomies for bronchiectasis can now be counted in the hundreds. This means at least 85% cures in these cases, and the operative mortality is now definitely within reasonable limits. In very recent years the dissection of the hilar structures and individual ligation of them has to some extent displaced the lung tourniquet, but it still remans a very important contribution to the progress of surgery of the lung.

Pulmonary Tuberculosis

In discussing the progress of the treatment of pulmonary tuberculosis one can divide it into two periods, that before fifty years ago, and that since. In the first period any treatment of pulmonary tuberculosis was feeble and inadequate. Sanatorium treatment had been proposed but it had not yet been adopted in many areas, and there was practically no surgery done. There had been valiant attempts. Tuberculous cavities had been incised and drained by Willis as early as 1676, Barry in 1727, Potau in 1783, and Godlee in 1887. Excision of tuberculosis lung was attempted by Block in 1881, and Tuffier about the same time, and the first successful lobectomy was performed by MacEwen in 1897 for secondary infected tuberculosis. There was no collapse therapy in this early period, although the foundations of modern pulmonary compression were laid as early as 1821 by James Carson of Liverpool, who urged the use of artificial pneumothorax as a means of collapsing cavities or controlling haemorrhage. He apparently had little influence at the time, because it was not until Forlanini of Pavia proposed and practised it in 1882 that artificial pneumothorax was really accepted as a therapeutic agent.

The past fifty years have seen the development of these two methods of treating pulmonary tuberculosis, that is, sanatorium treatment and collapse therapy, and they are now pretty much taken for granted. Artificial pneumothorax has become the commonest surgical procedure in the treatment of the disease, and is well-established, but there are still a great many cases for which this is not suitable or cannot be done, and there are also some problems such as the necessary duration of the pneumothorax which have yet to be solved.

The paralysing of the diaphragm by operations on the phrenic nerve was first introduced by Stuerz in 1911. This was done to provide aded rest to that side or to assist pneumothorax in lung compression. At first a permanent paralysis was achieved by pulling out the phrenic nerve, but more recently a temporary paralysis by crushing has been found to be just as satisfactory in the treatment of the lung, and to have few late complications.

The thoracoscope was first used by Jacobeus in 1905, and in 1913 he cut small adhesions through it to allow more complete collapse of the lung. This intrapleural pneumonolysis has been a great assistance to artificial pneumothorax in lung compression. Extrapleural pneumonolysis was started much earlier, and has passed through many stages in its development. Introduced by Tuffier in 1891, it was first considered necessary to fill the cavity with something solid to maintain the compression. Schlange in

1907 packed the cavity with iodoform gauze, and Tuffier in 1910 used a free human fat graft. Other substances used were paraffin of various kinds, bone, muscle and rubber bags. Generally speaking they were unsatisfactory, and Nissen in 1931, and Monaldi in 1933 left the space as an extrapleural pneumothorax, requiring regular filling with air at intervals, just as the intrapleural pneumothorax. This has been widely accepted, and is now a recognized procedure in collapse therapy. It is more of an operation than intrapleural pneumothorax but it can give a more localized collapse, and it is much less extensive than thoracoplasty.

The development of plastic operations on the chest wall to collapse underlying tuberculous cavities has been very interesting, and has been confined to the past fifty years. The first thoracoplasty was done by De Cerenville in 1885, and although he seemed to have the right idea, and he was followed by many others such as Quincke, Spengler, Turban, Bier and Landerer in the next twenty years, their results were generally unsatisfactory. In the light of our present knowledge and experience, the reason for the poor results is obvious. The resections of ribs done by them were not extensive enough, and were not properly placed. This was recognized by Brauer and Friedrich, and in 1907 they attempted a much more radical resection. They resected first, the second to ninth ribs, along with periosteum and intercostal muscles, then later the first to tenth ribs. This extensive operation was found to be very dangerous, so by 1909 they suggested a two-stage operation leaving the periosteum and intercostal muscles, and confining the resection to the upper ribs. This seems to be very similar to the modern thoracoplasty, but the development of the operation had yet to go through the stage of the paravertebral resection. This was introduced by Wilms in 1911, and consisted of the removal of three to four centimetres at the vertebral end of ribs one to eight, including the tip of the transverse process. This was modified many times by Wilms and Sauerbruch in the next fifteen years. Sauerbruch holds a unique place in the history of the surgery of pulmonary tuberculosis, and had a very great influence in its development. He was one of the first to set down specifically the indications and contra-indications for surgical treatment. The modern operation which is used most commonly now is a multiple stage resection localized to the area requiring collapse and involving removal of all the first and second ribs, and a gradually lessening amount of each successive rib. The transverse processes are removed if complete lateral collapse is required and the anterior remnants of ribs three to seven may be removed through an anterior stage if very extensive collapse is necessary.

The discussion of the surgical treatment of tuberculosis cannot be considered complete without reference again to one of the earliest surgical procedures attempted. The lobectomy by MacEwen in 1897 was mentioned and it is quite obvious why that operation did not become popular. At that time, lobectomy was a very dangerous procedure, and little hope of survival could be offered to the patient. With improvements in technique in both surgery and anesthesia, the operation of lobectomy has become quite common, and it can certainly be applied to the treatment of pulmonary tuberculosis. At the present time, radical excision has been limited to tuberculosis complicated by bronchial stenosis, bronchiectasis, or unusual cavitation.

Neoplasms

As in most other areas of human anatomy, the superficial and obvious diseases are usually the ones diagnosed and treated first, so in the realm of chest surgery the tumors arising in the chest wall were the first to be excised. Kronlein reported the removal of a sarcoma of chest wall in 1883, and it was the first of many such reports. Such a removal, if extensive, was a very dangerous operation in the days before positive pressure anesthesia, and was justified only because of the seriousness of the disease. Sauerbruch introduced the negative pressure chamber in 1903, which considerably improved the operative mortality, and then the introduction of positive pressure anesthesia fairly recently was of further assistance.

Much more dramatic, and actually of much greater importance was the treatment of carcinoma of the lung by radical excision. As early as 1891, Tuffier had performed experiments on animals removing lobes of the lung, or the whole of one lung. These experiments were very important, but it was a long time before their technique and results were successfully applied to man. As mentioned, Körte performed the first successful lobectomy in man for bronchiectasis in 1907, but it was not until 1931, that Nissen first successfully removed the whole lung, again for bronchiectasis. Carcinoma of the lung was first successfully treated by pneumonectomy by Evarts Graham in 1933. He reported this in Toronto in the Balfour Lecture of 1935, the patient still alive and well. Since that time successful pneumonectomies for bronchiogenic carcinoma have become quite common, and survivals of up to eight and nine years have been reported. These could quite easily be considered cures, and of a disease which for so many years and until recently was considered definitely incurable.

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FIRST YEAR IN RETROSPECT

DORCAS M. BEATON, '47

Two months of our second year in medicine have now passed, so that it is possible to compare last year's curriculum with this year's and to make a few interesting observations. On looking back it is possible to pick out certain good features of the present course and also to comment on others which, if improved, would be a great help to the first year students.

On reviewing the first year work in chemistry and physics, one notices immediately the similarity they bear to the upper school courses in these subjects. At present this is a necessity as so many students enter medicine without these upper school sciences, but when one considers the additional work this lack entails, one wonders why the students should be handicapped in this fashion. Wouldn't it be better to list chemistry and physics as requirements for entrance into first year? Those students who instead of the two physical sciences took the fifth form biology, whilst handicapped in two subjects, find themselves repeating, to some extent high school biology, although from a rather new angle. University biology differs more from the high school work than do the other sciences, and hence entails a set amount of studying despite the student's previous grounding. The net result is that a student who choose the biology option in high school is more seriously handicapped than he realizes. Chemistry and physics are not difficult and the student who already has had experience along those lines, has a much better chance of doing well in his first Medicine. Why should not physics, chemistry and biology all be pre-requisite subjects for entrance to Medicine?

Then on looking at the pass subjects one notices exactly the opposite attitude manifest towards them as towards the scientific ones. In Medicine the latter are far more important but still when an effort is made to give the students a chance in more cultural fields, a higher standing along those lines should be expected of them. A science course is liable to become too one-sided, and when one considers that a doctor is supposed to be a leader in his community and also the fact that during his student days he hasn't a great deal of time to devote to other than medical subjects or subjects on his curriculum, one realizes the value of first year pass subjects. Other universities demand a B.A. of anyone registering in Medicine, showing the importance they attach to the Art's subjects.

Another feature that is liable to make first year difficult is the method of teaching, and the great jump between high school and university. Here, the students who until recently were copying their notes off the classroom blackboard, have to make lecture notes and to distinguish for themselves the important from the unimportant. Some students adapt themselves to these new tactics readily whereas others take reams of notes and hence have to spend study periods distinguishing the various points and remaking their notes; a few miss the point of the lecture completely. But it is to be noted that in second year medicine the curriculum resorts once again to spoon-feeding methods. In anatomy, for example, the important headings in each lecture are outlined at the front; in chemistry, the notes are

written on the board; in histology, a set of typed notes are distributed. Wouldn't it then be possible to aid the first year Meds and to teach them more by methods they are accustomed to, if in second year this is going to be the case?

Biology seems to be the most difficult subject of the curriculum though. Statistics, one hears, have shown that it is the best testing ground to demonstrate what success the undergraduates are going to have in their upper years, but even so a few changes would simplify the subject considerably. One of the biggest problems is to know exactly what text book to use. Borradaille isn't sufficient, and hence it is necessary to become familiar with a long list of reference books. Wouldn't it be possible to have one text book that covers the course? At present Walters, Little, and Neil and Rand, are all fairly popular, but even so none of them touch on all the features of the course.

Then, too, one looks at the subject matter of Biology, and notices that the first term is spent doing the anatomy of the rabbit, and the last term reviewing the different orders of invertebrates and vertebrates. Wouldn't it be a better idea to study comparative anatomy in the first two months of the term, for this after all is more or less a review of fifth form work, and then devote the latter half to a study of the functions and relations between the nine organ systems, and the anatomy of mammals. This then would give the students a slightly better grounding in physiology, histology, and anatomy, provided these last three features are emphasized.

Biology, chemistry and physics though are but a stepping stone to far more interesting subjects, and this seems to continue from year to year, each year being a little more fascinating than the previous one. First year at the time seemed to be fairly difficult, but in retrospect it wasn't the curriculum that made it so, but the students' methods of attacking it, and in some cases a lack of proper grounding. Grounding is important and this fact has been borne out by the medical aptitude tests, which have shown that in many cases a low mark in this test indicated dissatisfaction after graduation, or a great deal of difficulty achieving one's aim.

ACADEMIC FREEDOM

TED KOBER

When embarking on a university career the first-year student, eager as he is and still unspoilt by disillusions, passes through his term blindly, with a complete lack of criticism of the methods of teaching and learning that are offered to him. It is only later on that he realizes the faults inherent in our present educational system, and it is only then that he voices his opinion on how this system could be improved, on methods which he believes will ultimately lead to the desired goal: perfected education. But it is also then that he, in his youthful or even, sometimes, infantile desire to reform, fails to see the value of many of our institutions. He arrives at the stage where he criticizes blindly, accuses everything and everybody of being useless and out of place. For now, therefore, our purpose is to show the unwarranted critic how fortunate he is, still able to enjoy the academic freedom which is denied his fellow-students and teachers in the less blessed countries on the European continent. We shall attempt to illustrate our case, using Germany as an example, by a short historical review.

European and especially German universities were proud of the centuries of tradition behind them. Only the lucky individual who has spent days wandering through time-honoured lecture halls and auditoria actually realizes the immense value of tradition, especially of a tradition that was constantly improved by various types of progressive innovations. The German student, by Canadian and American standards, could enjoy all the freedom he desired. Never was he subject to enforced lecture attendance or a set curriculum. All that was required was that he prove his wealth of knowledge at the final examination. He was free to choose his own teachers, who, by the way, were salaried according to the number of students attending their lectures. This measure in itself gave the guarantee that only teachers of outstanding quality were able to retain their office. Although the number of scholarships offered just about equalled that on this continent, it was usual for the student to spend his time freely at some outside occupation, to which he attended even during the academic session. Science was taught and practiced for the sake of science only, and any social status which might accompany the respective profession was merely a secondary consideration. The student was encouraged to take an active part in political life, an opportunity of which many freely availed themselves. High degrees of specialization, so commonly encountered on this continent, were only rare results of routine German university education.

The attitude towards academic freedom was drastically changed with the advent to power of the Hitler regime. The Nazi view can best be summarized by quoting one of Hitler's remarks as made to Herrmann Rauschning: "Universal education is the most corroding and distintegrating poison that liberalism has ever invented for its own destruction."

In the light of this fantastic (but factual) emotional outburst it is easy to explain the changes that occurred in Germany after January 30, 1933. The social

and economic background of most Nazi party leaders incited their hate towards all striving for an intellectual betterment of the German multitude. The new educational system saw only one asset in post-high school training, and that was to view the university as a centre for producing highly skilled technicians who would employ their nefarious knowledge in the establishment of German world supremacy. The logical result was rapid decay of all German centres of higher learning. Coerced by necessity, the courses had to be abbreviated in order to assure a sufficient quantity of trained men in time for the preparation for and the actual outbreak of the Second World War. One of the outstanding features of the Nazified "educational" system was a wave of expulsions of great liberal thinkers and scientists, although in certain cases a voluntary exit preceded the official edict. This in turn necessitated the procuration of replacements for depleted university staffs, and it is hardly difficult to understand why these substitutes were inferior in quality to those teachers who had helped to make German education one of the greatest in the world. Above all the Hitler movement encouraged physical training in preference to education of the mind, and thus stifled the prospective student's desire for learning.

It would have more than sufficed had Hitler abolished only the German centres of education, but with typical Prussian thoroughness the recently acquired knowledge of destruction was immediately applied to occupied countries. Institutions of higher learning all over Hitler-dominated Europe are either functioning on part-time, or are entirely closed, although Germany has not everywhere been as radical as she was in the case of the University of Prague where students had to forfeit their lives.

The logical question to follow is naturally: "Well, what of all that? What can we, here in Canada or in the United States, do about the decadence of education in Europe?" Indeed, there is a great deal that we can achieve. This continent has, by its own progress as much as by necessity, become the center of learning for generations to come. We have the solemn duty to preserve all the traditions that enemy forces have brutally attempted to eradicate. We should learn a great deal from those men, who, either voluntarily or forcibly, have left the continent and come to our shores; we can acquire from them actual knowledge, while they can also teach us, because of their unfortunate experiences, how to prevent a similar spiritual decay in the New World. But our most important task will be to render active assistance in the process of European reconstruction, which is bound to follow the conclusion of the war. *Remember*, we, the students of today, will have to make and master the world in which we and our children are to live tomorrow.

Abstracts

G. G. CAUDWELL, Editor

Contributors { P. H. Spohn, D. B. Stark, G. J. Quigley

The Treatment of Acute Appendicitis in Children as Influenced by Chemotherapy

By Carl A. Wattenberg, M.D., and Peter Heinbecker, M.D.
Surgery, Oct., 1942

This paper covers the results of four years' treatment in the application of chemotherpy in appendicitis and its complications carried out at the St. Louis Children's Hospital.

Chemotherapeutic drugs are administered prior to operation in all cases of appendicitis where peritonitis is diagnosed or even suspected.

Dosage. The blood levels effective for sulphanilamide range from 8-14 mg. percent, for sulphapyridine and sulphathiazole 6-12 mg. percent. To reach a level quickly 1.0 percent sulphanilamide in normal saline or 5 percent of the sod. salts of sulfapyridine or sulphathiazole is given intravenously. They are all administered on the basis of 0.2 grm. per kg. body weight in six divided doses. Sod. sulfapyridine is given intravenously at the dosage of 1cc. of 5 percent solution per kg. body weight repeated every 8 hours; sod. sulphathiazole, 2 cc. per kg. body wt. Q6h. Occasionally 2-5 grams of sulphanilamide was placed locally in an abcessed area or in the area from which an adherent gangrenous appendix has been removed.

Complications from use of the drugs are not severe or frequent. The commoner ones are nausea and vomiting. There was no severe leucopenia or anemia. Cyanosis due to methemaglobin concentration is often encountered and is counteracted by administration of methylene blue.

The beneficial affects attributable to chemotherapy are: (1) Rapid lessening of the toxicity. (2) Rapid regression of the peritonitis. (3) Reduction of number of cases requiring drainage. (4) A shortening of the period of illness. The added affect has been to practically eliminate death as a sequel to peritonitis associated with appendicitis. Chemotherapy cannot be relied on alone and proper surgery still plays the most essential role.

Preoperatively electrolytic and nutritional deficiencies of the patient must be corrected. Ringer's solution, dextrose and plasma intravenously are all used preoperatively. The usual time to prepare the patient is 2-6 hours. Drainage is used only if abcess formation has injured the peritoneum badly, but not if the peritoneum is diffusely involved.

Postoperative chemotherapy is continued at a high level until the temperature subsides. Intravenous fluids are favored immediately after operation rather than the early administration of food by mouth. After the temperature subsides the drug is continued at one half the previous dosage for 3-5 days.

During the last four years there have been 223 appendectomies with five deaths, a mortality of 2.2 percent. No deaths

occurred following operations for acute unruptured appendicitis, recurrent unruptured appendicitis or acute appendicitis with local peritonitis. The five deaths resulted from appendicitis associated with diffuse peritonitis. During 1940 and 1941 there were no deaths and in this period the principles of treatment were employed unfailingly. The mortality from acute appendicitis and its complications has been reduced from previous average of 7.8 percent to 2.2 percent.

P. H. Spohn.

Subtotal Gastrectomy For Stenosing Duodenal Ulcer

A. W. Allen, M.D.
The J.A.M.A., Oct. 17, 1942

Although a more careful consideration is given to the stenosing duodenal ulcer, a brief summary of the other complications of duodenal ulcer, i.e., acute perforation, massive haemorrhage and intractibility is also presented.

(1) Acute Perforation: Early operation and simple closure is the procedure of choice. The earlier the closure is done, the lesser the risk, provided the perforation is under twelve hours duration. If passed this time limit when first seen, the question of spontaneous sealing and further conservative measures must be considered. Radical operations for acute perforation are contraindicated.

(2) Massive Haemorrhage: The summary of the data of the Massachusetts General Hospital reveals that nearly all patients under the age of 45 will spontaneously recover from acute bleeding while approximately 30 percent above this age will succumb. This variation is believed to be due largely to the inability of an arteriosclerotic vessel to contract sufficiently to hold in place a thrombus over a long enough period of time. Further, if operation is performed within 72 hours of the onset, success is likely. Thus the younger patient should be treated conservatively and when his optimal health has been regained, he should undergo a radical cure. The older patient should be evaluated and the decision made for or against radical treatment within 72 hours of the onset.

(3) Intractability: This term includes those cases which do not respond to conservative measures. Such are those which penetrate the pancreas, interfere with the normal function of the biliary tract or involve the pyloric sphincter. These patients seldom remain symptom free unless on a hospital regimen and quiescence of the ulcer is incompatible with work which so involves an economic factor. Because of the usual lack of cooperation with these patients, the highest percentage of failures is found here.

(4) Stenosing Duodenal Ulcer: Either because of repeated activity with extension in a circular direction or because of the development of multiple ulceration on opposite walls of the duodenum, a constriction from scar tissue is formed. It is usually found past middle life, in which there is found a prolonged ulcer history characterized by almost constant or recurrent signs of activity over this period. This finally becomes a mechanical problem and the patient seeks help because he can no longer absorb enough nourishment to maintain life.

Subtotal Gastrectomy is now believed to be the procedure of choice, in favour of the less adequate palliative operation such as gastro-enterostomy in which a jejunal ulcer frequently appears. Further, Subtotal Gastrectomy for Exclusion, in which the antral mucosa was left in situ, but extensive fundusectomy done, an-

astomotic ulceration was also very high in incidence. In the radical procedure recommended, the best results were gained in those cases in which the distal half or more of the stomach was removed together with the antrum, pylorus, and ulcer bearing portion of the duodenum.

G.G.C.

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Carcinoma of the Prostate

Wm. C. Quinby, M.D.

Carcinoma of the prostate is quite common and from a clinical viewpoint about 20 percent of males over 50 years who have symptoms of prostatism, are found to be due to a prostatic carcinoma. Indeed post-mortem examination brings to light additional cases of carcinoma in patients who showed no evidence of prostatic carcinoma in life. The incidence increases with age until it reaches about 30 percent in patients in the ninth decade.

So far as the point of origin is concerned, the lateral and posterior lobes are most frequently involved, the neoplasm springing from cells which had previously undergone atrophy of a senile type. The growth never arose from the Median lobe and in several cases appeared to be multicentric. The tumor cell was squamous in 3 percent of cases and adenocarcinoma in the remainder. Of the tumors recognized clinically or grossly at necropsy, 54 percent had produced metastases, which reflects the clinical experience that prostatic cancer is a slow growing neoplasm which spreads most frequently by metastases rather than by invasion of contiguous structures,

The tumor is believed to spread by way of the perineural lymphatics which were found to contain malignant cells even in early stages of the disease. This method, together with transport in the blood stream, gives ready access to bone and explains the marked frequency in which the bony pelvis and the lower vertebrae become involved as compared with more distant regions.

However, the clinical evidence afforded by the recorded cures after total prostatectomy, together with the well-known slow progress of these tumors, suggests that this perineural invasion is of little prognostic significance and may represent only the terminal picture of the malignancy. Consequently the attitude of defeatism concerning operative intervention is unjustified.

The study of the acid phosphatase in blood serum is of value. In normal persons this was found to be from 0.5 to 2.5 units per 100 cc.. In patients with bone metastases from prostatic carcinoma a marked increase takes place, whereas in the sera of those with prostate cancer but no metastases, the acid phosphatase readings are normal.

The demonstration of the relation between the prostate and the sex hormones has broadened the field of therapy for carcinoma of the gland. The depression of prostatic secretion in both man and animals following castration is dependent upon the reaction of the male sex hormones, the androgens which is secreted by the testes. Consequently, with a view to depressing the activity of the epithelial cells of the prostatic carcinoma by a depression in the androgenic hormone, castration in 21 patients with advanced disease was studied. In the 18 patients who were improved, the most striking benefit was the relief of conditions caused by metastases with some degression in size of the primary growth. Other methods of reducing the androgens

in the body are, the exposure of the testes to the destructive action of the X-ray and the use of an oestrogen.

Total removal of the gland whenever possible is a generally accepted procedure and furnishes excellent results but where impossible because of carcinomatous invasion, resection of the obstruction through the urethra is of value. The use of radium either as a salt or as emanations in "seeds" is no longer popular.

G. G. C.

New England Journal of Medicine
Vol. 227, No. 14

Carcinoma of the Bronchus

George W. Holmes, M.D.

This presentation is based upon a review of the recent literature and a study of over 200 cases treated at the Massachusetts General Hospital. The discussion is limited to roentgenological findings in early cases and to their treatment with roentgen rays because only a very few patients present themselves at a time when their disease would lend itself to surgical treatment. In this series there were no 5-years cures, althought several cases have been reported, but this method offered much in the way of relief from symptoms and in many instances definite prolongation of life.

The disease, which is about as common as cancer of the rectum, progresses rapidly, the duration between the onset of symptoms to death being a matter of months rather than years. Consequently the delay of a month before instituting treatment may mean the difference between success and failure. The early symptoms and signs are not characteristic and may be absent. The late symptoms are those of its complications which usually indicate its incurability.

Roentgenologic examination often gives the first clue to diagnosis, which with the clinical findings, is established by bronchoscopy, thoracoscopy or surgical exploration. The type of tumor as determined from a biopsy taken at bronchoscopy is of importance in determining the form of treatment to be followed, as oat cell and undifferentiated carcinomas are radiosensitive tumors and usually disappear locally under such treatment, whereas surgical results in these groups have been bad. Adenomas and slower growing types of malignant tumors are radioresistant but give excellent results when treated surgically. Advanced cases with lung necrosis or empyema are made worse by intensive irradiation and should be treated surgically if at all.

Histological classification of the disease showed epidermoid carcinoma in the majority of instances, followed by oat-cell carcinoma, undifferentiated and adenocarcinoma in that order. Of the cases seen between Nov. 1940 and March 1942, 45 of the 68 were dead, 8 were living and the remainder could not be traced. Ten of this group were considered suitable for surgical treatment and of these but three are now living. In this same period 47 were selected for irrariation and of these 8 are now living, 2 being free from evidence of disease, 4 symptomatically improved and 2 were in the terminal stages of the disease. The average duration of life for those known to be dead, from the time diagnosis was established until death, was four and a half months.

A pathological interpretation of roentgenologic findings is given together with excellent schematic representations of such as is found on the films which most certainly warrants attention.

G. G. C.

Castration for Carcinoma of the Prostate

C. H. Neuswauger, M.D., and
Vincent Vermasten, M.D.

The New England Journal of Medicine,
Vol. 227, No. 17
Oct. 22, 1942

Since the report of the effects of castration on carcinoma of the prostate by Huggins et al. in June, 1941, these individuals have operated on some 41 cases and so present their observation and discussion in the form of four interesting case studies. These substantially confirmed the results of Huggins and his co-workers.

Of the 41 patients upon whom bilateral orchidectomies were performed, none regret having the operation done. On the average, the patients gained five lbs. in weight and in many instances as much as 20-30 lbs. The most striking changes observed were a marked increase in appetite, complete relief from pain, improvement in bowel habits and a feeling of well being. There was also complete relief of urinary symptoms although this was largely due to the transurethral resection of the disease which was done in 40 of these cases. In one instance there was a miraculous response which resulted in the alleviation of the symptoms of paraplegia from a metastatic lesion in the spine.

G. G. C.

Case Records of the Massachusetts General Hospital

Tracy B. Mallory, M.D., Editor
The New England Journal of Medicine,
Oct. 22, 1942
Case 28432

This should be of special interest to those in the final year and in particular to those present at the clinical-pathological conference of a recent date when an interesting case of an undiagnosed, unsuspected case of liver abscess was presented. A similar case, although differing in aetiology, is presented herein and discussed.

G. G. C.

Premonitory Symtems of Myocardial Infarction

Norman H. Boyer, M.D.

Since 1912 when Herrick first described the characteristic signs and symptoms associated with myocardial infarction, atypical cases have ben recognized with increasing frequency and indeed the treatment of this disease is generally understood and applied such that now attention is being focussed on the recognition of premonitory symptoms such that the instatution of therapeutic measures may diminish the severity of the illness or even prevent clinically recognizable infarction entirely.

In many cases, the onset of infarction is not sudden and unpredictable but is preceded for a variable time by premonitary symptoms which are presented and discussed in the form of case studies.

Sudden onset of angina pectoris, or the sudden increase in the severity and frequency of pre-existing angina pectoris or repeated attacks of prolonged chest pain without signs of cardiac infarction have been found to precede the onset of infarction in some patients. The recognition of these prodromal symptoms should be followed by rest, sedation avoidance of tobacco and the use of papaverine, which may reduce the size of the eventual infarction or forestall it entirely.

The enforcement of two to four weeks in bed for a patient in whom there is no

proof of impending infarction, or proof that the patient will benefit in any way from such, seems to be radical treatment. Balanced against the possible death of the patient from acute infarction, a prolonged illness extending from three months to one year or more, chronic invalidism or at best, a heart with a large myocardial scar, two or three weeks in bed, if there is the least hope of preventing these sequelae, seems conservative treatment.

G. G. C.

The Present Status of Cooling Limbs in Preparation for Surgical Procedures

Robert T. McElvenny
American Journal of Surgery, Oct., 1942.

Frederick Allen and his co-workers have shown that limbs after cooling may be amputated without any anaesthesia. He writes, with this method, the operation was less shocking, the mortality rate lower and the results as satisfactory as any other method.

In preparing a case for amputation, the following steps are taken: a tourniquet is applied to the limb high above the site of amputation; the limb is then immersed in ice water or is surrounded with shaved ice to just above the site of amputation; then after one to three hours the limb may be amputated at the site of election. The patient experiences no pain, the field is bloodless and the shock much reduced. The procedure has been used in those cases which are a poor risk, i.e. diabetic or arteriosclerotic gangrene.

In this paper, case records are presented which demonstrate clinically that cooling a limb and so decreasing the circulation, will prevent the absorption of toxins, the advance of bacterial infections and the relief of pain. D. B. S.

A New Approach to the Diagnosis of Herniation of the Intervertebral Disc

Wm. Duncan and Thomas Horn
S. G. and O., Aug., 1942.

The pathological anatomy of the herniated disc is discussed.

Although the traumatic neuritis caused by disc protrusion is often the most prominent symptom of this condition, the neurological findings cannot be relied upon to localize the lesion. In two cases with identical neurological signs, the lesion may be located in two different areas. However, certain relationships between neurological findings and those seen at operation are worthy of note: Bilateral neurological findings, with rectal and vesicle disturbances, suggest a midline lesion. Unilateral involvement of more than one dermatome also indicates a more centrally located lesion. Unilateral involvement, consistently confined to one segment, indicates a lateral lesion. The disturbance of joint mobility is also used to localize the lesion in so far as limitation of movement and attitude are concerned.

The intensity of peripheral pain or paraesthesia is proportional to the force of the herniation against the involved roots which in turn is proportional to the degree of compression of the disc by the vertebral. Thus to reduce this, there is a movement of the spine away from the side of the lesion, and as the extrusion is posterior, an attitude of forward flexion is assumed.

By taking spinal bending films of the patient in lateral flexion and in forward flexion and extension, decreased mobility may be visualized at the involved intervertebral joints by the failure of the joint space to narrow or allow extension when

compared with the joints above and below this area. These films, however, serve only to establish the level of the extrusion rather than its presence.

A procedure to demonstrate pain on hyperextension is described—the patient is supine, and the knees are allowed to flex over the end of the examining table, thus producing a definite increase in the Lumbar Lordosis. The sacral and lumbar roots are thus completely relaxed due to the flexion of knees and extension of the hips. Consequently any pain from these segments must arise from pressure against rather than traction on the nerve roots. Finally, flexion of the limbs, by decreasing the lumbar lordosis, affords complete relief of pain. D. B. S.

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PHOTOGRAPHS

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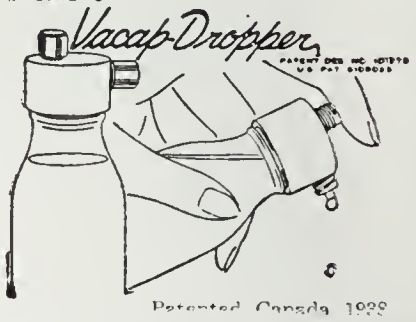

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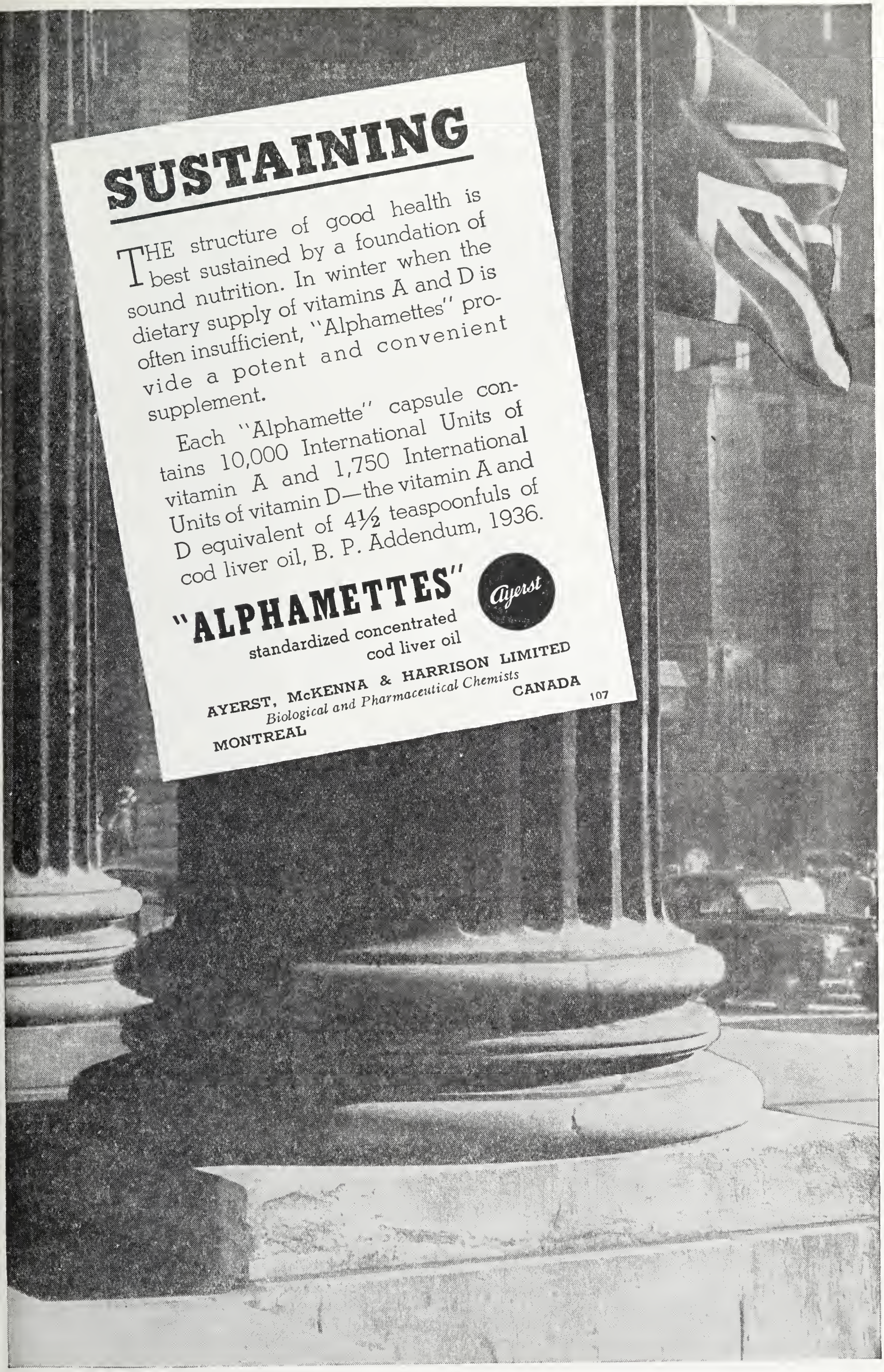
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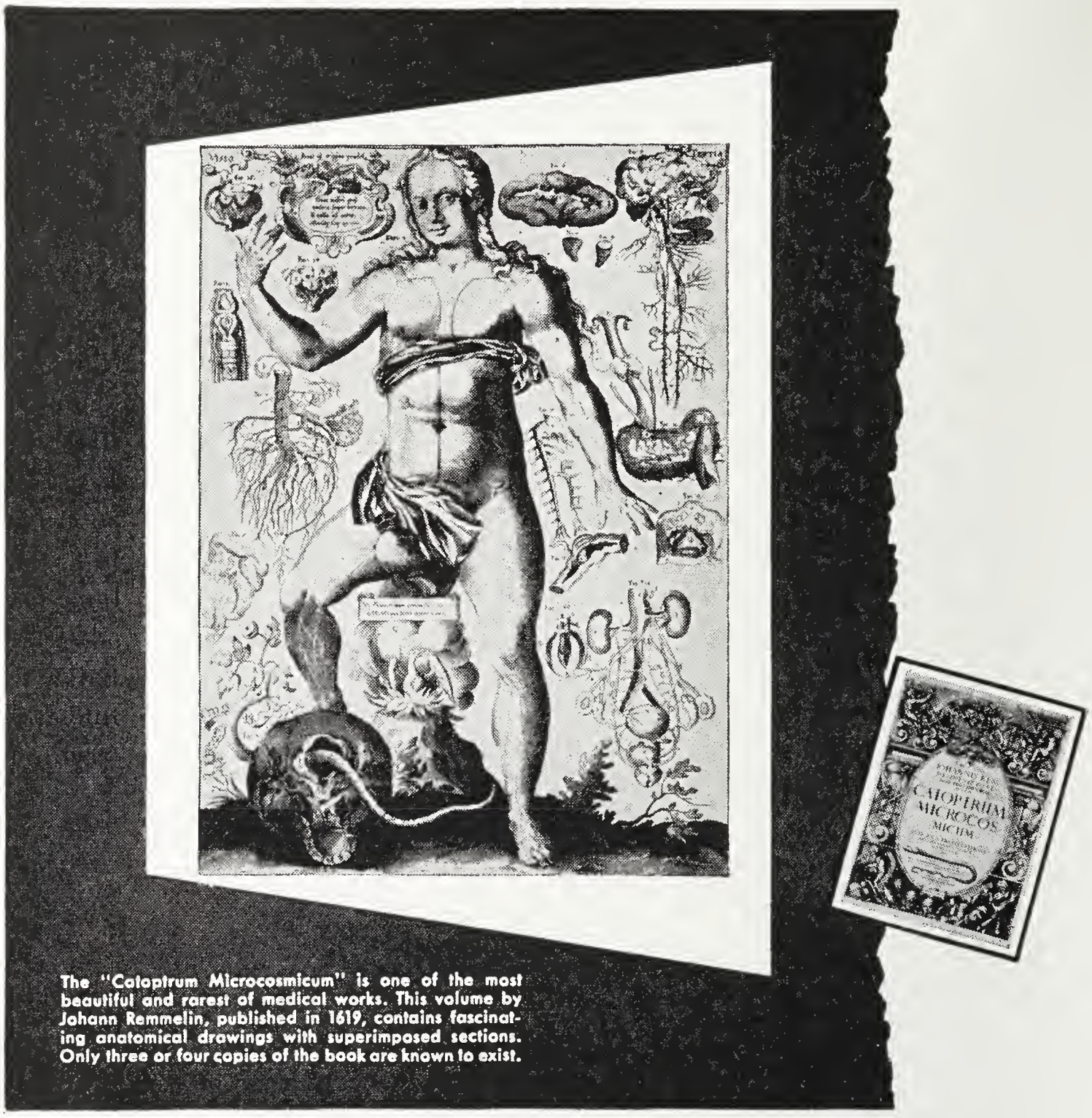

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(1) 1939. Food and Life; Yearbook of Agriculture, U. S. Dept. Agriculture, U. S. Gov't. Printing Office— Washington, D. C.

1940. J. Am. Med. Assn. 114, 548

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(1) 1938, Nutrition Abstracts and Reviews 8, 281
(2) 1939, Food and Life: Yearbook of Agriculture U. S. Dept. Agriculture, U. S. Gov't Printing Office, Washington, D. C.

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